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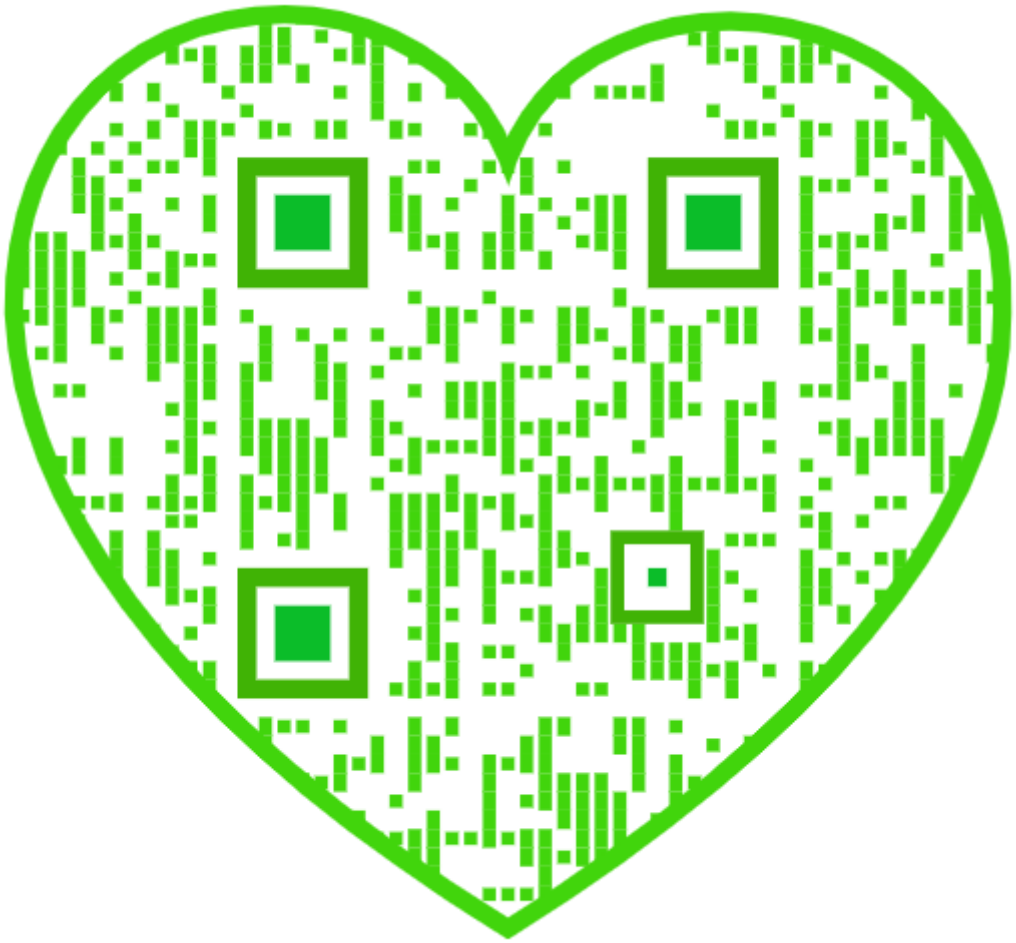

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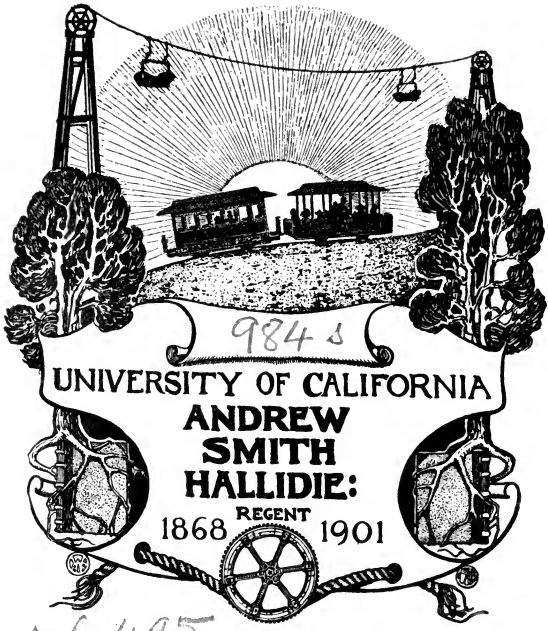

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DISEASES OF THE HEART:

THEIR

DIAGNOSIS AND TREATMENT.

BY

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TO

WILLIAM HENRY PATTERSON, ESQ.,

of San Francisco, I dedicate this volume, as a token of admiration and long personal friendship.

DAVID WOOSTER.

14 Geary street, San Francisco,
September, 1867.

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PREFACE.

In writing the following pages, I have been scrupulously careful not to place my own unsustained assertions in opposition to the results of clinical demonstrations or extreme probabilities. I have also endeavored to give the anatomy, or topography, of the structures under consideration, with as much accuracy as possible.

There are no such obstacles now, in the way of writing intelligibly on diagnosis of diseases of the heart, as there were even twenty-five years ago. If medicine has not yet become a science, it has, at least, so far advanced as to recognize its own defects, and to be conscious of its absolute knowledge. Physicians now know where their knowledge ends and their theories begin; and in this they are far in advance of their forefathers.

My aim has been to limit myself, as closely as possible, to known facts, and to indulge in no idle speculations, for the purpose of introducing some favorite theory of my own, in order to make myself seem original. I need not tell my intelligent readers that this treatise is merely an abstract; but it is hoped that they will find it what the author designed it to be, a careful and conscientious epitome of the knowledge now possessed by the profession on the subject of which it treats. My only claim to their commendation will consist in having placed the knowledge already possessed in a more accessible form, of having made the facts we daily need to use more tangible, by stripping them of some of their garniture, and grouping them in closer and more obvious relations to each other.

I have endeavored to condense the statement of facts into a much smaller compass than they appear in the standard authors, without omitting anything necessary to a clear understanding of the subject treated. In the process of condensation, I have frequently used several successive words, just as they occurred in the authority consulted, and perhaps occasionally a full sentence, without quotation marks. But, while I make no pretence to having written a work original in material, I have endeavored to avoid the crime of plagiarism.

I have ventured to make some suggestions in diagnosis, not of a radical kind, but merely as to applicability and import of certain signs, and also to indicate additional treatment in some forms of heart disease to that which authors generally recommend; but, as I have at the same time pointed out wherein I have ventured to differ with the masters, the reader will not be beguiled into mistaking my treatment for that of authors whom he may with just reason hold in higher estimation. I have insisted with some pertinacity on the reflex treatment of unsoundness of the heart, but for my opinions on the value of this kind of treatment, I am indebted to Radcliffe, Brown-Sequard, and others who have investigated the more recondite departments of human physiology.

I have had much less to say of treatment than of diagnosis, because in reality much less is known of the former than of the latter. We are yet entirely ignorant of the manner in which, or of the prime cause why the heart becomes unsound by the deposition of analogous tissues, in most cases, and until we have acquired this knowledge, we cannot do much more than treat certain symptoms, which are common to either acute or chronic inflammations. But thanks to our almost exact knowledge of the beginning and process of acute inflammations, derived from a multitude of clinical

cases, carefully observed in all countries, by eminent and conscientious physicians, we are now able to treat symptoms with a degree of rationality which will accomplish a purpose predetermined on the part of the physician, and which will generally result in success in curable cases, and in the alleviation of the most hopeless.

Cases of heart disease are of alarming frequency in California, yet, in the absence of statistics, it would be improper to say categorically that this affection is excessively frequent here; still I feel convinced that it is, and were its etiology within the scope of this treatise, I think it could be satisfactorily shown why disease of the heart is more likely to be prevalent in newer than in older civilizations, and in this climate than in many others; and under the special, moral, mental and hygienic influences which characterize us, than under those which predominate in other centres of population. For example: the physical aspect of the country and metropolis—mountainous and hilly—reverse of fortune and the vital depression consequent upon it; family ties rudely sundered, and the despair resulting; the insatiable thirst for riches, and the unusual energy employed in acquiring them, to the deprivation of mind and body of essential rest; the exposure, night and day, to which our mining population is subject; the tunnel and deep drift work of the quartz miner; the excessive intemperance in drink—the thousand disappointments, and misfortunes, and endless unrest to which a population anxious to become rapidly rich, like ours, must ever suffer, are so many reasons why the heart should become diseased and exhausted under the great labor of providing for such extravagant use of all the functions of life.

It is on account of my opportunities for observation during the seventeen years I have been practising medicine in California, and the hope that I might add some little to the facility of diagnosis, and the success in the treatment of

affections of the heart, and that I might materially diminish the labor of finding the knowledge that has already been published, that I determined to make this slight addition to medical literature.

This small work has been prepared during the intervals of daily toil, and hence it lacks that unity of design and uninterrupted sequence of subjects and sentences which are desirable in a strictly systematic treatise; but inasmuch as it does not aspire to that category, I trust it will be considered with some indulgence in this respect. Those who desire a complete, systematic and exhaustive treatise, written with the most logical clearness, will find that of Walshe on "Diseases of the Heart" unsurpassed in any language.

I have freely consulted Walshe, Hope, Latham, Stokes, Bennett, Markham, Skoda, Rokitansky, Radcliffe, Brown-Sequard, See, Virchow, Claude Bernard, Flint, Piorry, and numerous other authors; also, current medical literature of standard authority.

I am under many obligations to my friend Dr. J. N. Brown, of San Josè, one of the most learned and successful physicians in California, for valuable suggestions and criticisms during the compilation of this epitome. I am conscious of many imperfections in it, both in diction and manner of arrangement. With many misgivings, I offer it to the profession, asking for it merely the indulgence which a conscientious author has a reasonable right to expect of a conscientious reader.

DAVID WOOSTER.

San Francisco, September, 1867.

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CHAPTER I.

INTRODUCTION. CAUSES OF DISCREPANCIES OF OPINION IN CASES WHERE EXACTNESS IS POSSIBLE.

1. Many works have been written on diseases of the heart, and its affections have been described and treatment recommended in all standard works on the practice of medicine; but it has occurred to me that the matter might be much condensed, without detriment to scientific exactness and with great benefit to actual sufferers.

2. Physicians well know in their own minds that a very small proportion of the practitioners of medicine are able to diagnose an affection of the heart with even tolerable exactness; not only are they unable to say what valve is affected, but they do not distinguish the side of the heart; still worse, many who listen cannot distinguish normal from abnormal sounds, the beat of a healthy from the throb of a diseased heart.

This inability of discriminating sounds, with some is a natural defect, a physiological defi-

ciency in the ability to compare small discrepancies. But the inability generally is less serious; it is a lack of the acquirement and recollection of facts which may be acquired and remembered by most persons of intelligence. These facts can be acquired only by diligent study, and numerous examples, carefully and rigidly observed. For example: a person who has acquired the elements of Latin, when he attempts to translate a sentence of Latin into English, if he is really acquainted with the structure of the original, is altogether sure when he has rendered its true meaning. If he have less or more than the true meaning, he will be conscious of the deficiency or redundancy; or if different from the true he will know wherein the difference consists. But one who lacks this knowledge of the principles of the original language, will never be quite sure of his translation; there will always remain some doubt in his own mind about the exactness of his rendering. No amount of labor, without a knowledge of the fundamental facts, will be of any avail. So he who practices physical diagnosis, must first of all, be well grounded in the meaning of natural sounds and signs, and in

their topography on and in the body. Of what use is it to recognize *crepitus* if we do not know what *crepitus* indicates. Of what use to recognize murmurs if we know not from whence they proceed, their cause and significance? Two physicians, with diplomas from respectable medical colleges, will examine a chest the same day. One will say there are softened tubercles in a lobe of the right lung, but that the left lung is natural; the other will say the right lung is not affected at all, but he finds one or more cavities in the left lung. Now, if the patient goes on to other physcians, perhaps the next will tell him he has a bronchitis or a catarrh; the third will tell him his "lungs are more than half gone," while a fourth will tell him categorically, his lungs are perfectly sound, but that he has disease of the heart.

All physicians recognize this gamut of opinions on the same case, examined the same week. Whence do such discrepancies arise? They do not always arise from inability on the part of the examiner, less frequently from lack of frankness. Yet the result is the same on the mind of the patient and his friends and of the community, as if the disagreement in opinion were

the result of ignorance. In some cases, doubtless in most, when the discrepancy is so irreconcilable, the doctors have examined superficially, and the ear of each has been placed over a different portion of lung, and the examination has not been minute and exhaustive. One listened, perhaps, at a lower lobe, the other at an upper, one placed his ear just below a clavicle, the other placed his just behind a scapula; one listened carefully on one and carelessly on the other side, while the other observed closely both sides of the chest; and finally one knew not the meaning of the sounds elicited, or heard with the ear, while the other appreciated their precise significance.

But until professional opinions shall more nearly harmonize on matters of simple fact in physical diagnosis, the people will not have *implicit* confidence in the opinion of any one of us, however excellent.

If a patient with aortic insufficiency, or tubercular cavity under the right clavicle, should be examined by twenty physicians in succession, and the whole twenty should verify the insufficiency in the same place, or the cavity in the same portion of lung, and if similar harmony

of opinion on matters within the reach of exact diagnosis, should prevail even with twenty physicians in one large city, where there are five hundred doctors, as there are in San Francisco, I say, if twenty would agree on ascertainable facts, the profession would very soon possess the entire confidence of the people. The fault is in our own carelessness or inability; and whether we are careless or ignorant, the result is much the same.

4. I purpose in the following pages to give a brief summary of what is *known* about diagnosis and treatment of diseases of the *heart*, and to show how simple it is for all educated physicians to say positively what portion of the heart is affected in most cases, and to show in the few cases in which positive diagnosis is unattainable that an extremely probable diagnosis may be given, sufficiently exact at least to form the basis of rational treatment.

CHAPTER II.

ANATOMICAL AND PHYSIOLOGICAL FACTS CONCERNING LOCATION OF VALVES, AND ORIGIN OF HEART'S SOUNDS.

1. No two hearts are the same size. No two persons' hearts beat precisely alike, or so nearly alike, but that a practiced ear would distinguish the difference in the dark. Yet the healthy beat of the heart is so different from the unhealthy, that the practiced listener would be able to say without seeing the patient's face, whether the heart was healthy or affected.

2. The heart has a more or less conical form, and is suspended obliquely in the chest; its base corresponds with the median line of the breast bone, but is directed upwards and backwards towards the backbone, while its apex is directed downwards and forwards and to the left, so that when the heart contracts the apex is felt and seen to strike the chest between the fifth and sixth ribs, below and a little to the right of the left nipple, *when the person stands erect.* If he lean forward the beat strikes not only the fifth intercostal space, but the fifth rib and a little farther towards the breastbone. If he lie down

on his back, the apex beat is imperceptible anywhere. If he turn on his left side he renders it more perceptible than in the erect position, but in a larger space more towards the nipple than the sternum. And, finally, if he turn on his right side, the heart's impulse against the chest is scarcely, or not at all perceptible.

These are obvious facts, but well worth remembering.

3. Aside from all nice distinctions, the ear recognizes *two* natural sounds of the heart. These are known as the *first* and *second* sounds of the heart.

4. The first sound is heard most distinctly over the anterior surface of the heart, on the fifth costal cartilage, left side, at a point midway between its junction with the sternum and its junction with the rib. "It is long, dull, and smothered in tone, and occupies one-half the duration of a single beat." It corresponds in time with the impulse of the heart in the precordial region.

5. The precordial region is certainly found by describing a circle, with a radius of one inch, from a point in the fifth costal cartilage, left

side, midway between its junction with the rib and its junction with the sternum. "In this case we suppose a well formed chest." (LATHAM.)

6. The *second* sound is heard most distinctly in the situation of the aortic and pulmonic valves; it follows immediately upon the *first* sound, with scarcely an appreciable interval. It is short, sharp, and distinct in tone, and occupies one quarter of the whole of a pulsation.

7. "A line drawn through the inferior margins of the third rib, across the sternum, passes through the pulmonic valves a little to the left of the mesial line, and those of the aortia lie behind them, but about half an inch lower down." (HOPE.)

"The aortic and pulmonary valves are situated under the sternum, at the level of the third costal cartilage." (DALTON.)

8. The first sound of the heart is caused by the closure of the auriculo-ventricular valves—mitral and tricuspid valves—valves between the left ventricle and auricle, mitral; valves between the right ventricle and auricle tricuspid.

9. "A horizontal line drawn through ("along?" LATHAM) the under edge of the sterno-costal

articulations of the fourth ribs will cut across nearly the middle of the length of the mitral valve, when drawn outwards and downwards by its tendinous cords and fleshy columns, and pass about two or three lines above that portion of the tricuspid which most nearly approaches it, the latter valve lying underneath the sternum, and the former immediately to its left." (JOY.)

10. The *second* sound is undoubtedly caused by the closure of the semi-lunar valves (aortic and pulmonic). The *first* and *second* sounds occur in the same order as the closure of the two sets of valves: 1st, the two auriculo-ventricular valves—mitral and tricuspid, shut back, *first* sound; directly upon this the two sets of semi-lunar valves, aortic and pulmonic, shut back; *second* sound.

11. The sounds of the heart, their cause, and the location from whence they proceed being granted, and their quality being ascertained from the examination of persons in health, the inference is clear that any marked variation from this standard will be abnormal, and a sign, considered by itself, of disease of the particular

structures from which the marked variation proceeds.

12. Persons die of "heart disease" almost daily, but whether of disease of the inside or outside of the heart, whether of the valves or of the substance of the heart, the record scarcely ever shows. A distinguished London physician said, after thirty years of practice, that two-thirds of his professional life passed before he could discriminate between an inflammation of the cavities of the heart and of its outside; before he could tell an endocardial from an exocardial murmur. No physician in the world could make this distinction before 1826. And yet it is of great importance to know there is a distinction, and to know what the distinction suggests of the health and probabilities of life to the patient.

a. We will say then categorically, that an educated ear can, in almost every, if not every case, distinguish an *exocardial* from an *endocardial* murmur; and hence a pericardial from an endocardial affection.

b. Perhaps in nine-tenths of the cases in which the heart is diseased, its inside rather than out-

side is affected. Affections of the outside of the heart, or of the pericardium are comparatively rare.

c. Men are more liable to heart disease than women; adults are more liable than youths and children; residents of cities are more liable than residents of the country; dissipated people are more liable than those of regular habits. The rich are more liable than the poor.

d. Courtezans rarely have disease of the heart!

e. Excessive indulgence of the erotic passion, is, on the contrary, in men a great aid to the development of endocardial disease, and also of acute rheumatism, which is a conspicuous and very common forerunner of pericarditis.

f. It is obvious there is no opposition in propositions *d* and *e*, but rather correlation. There is all the difference conceivable in the physiological effect of erotism in man and woman.

13. It may be said in general terms that every *acute* disease of the heart is an inflammation, and if rationally treated, does not necessarily cause death.

But the world is much more concerned with

those diseases of the heart which are not inflammation, and that do not result from any direct injury, or any sudden or acute disease.

Certain growths in the body are called *heterelogous* as tubercle, carcinoma, cephaloma, etc., and the heart even is not exempt from heterologus growths of this nature; but they are exceedingly rare, so much so as to be almost beyond the field of research.

But permanent disease of the heart is derived from the deposition in some portion of it, of analogous tissues; that is analogous to healthy tissues. The cartilage and bone deposited in the heart and arteries is analogous to cartilage and bone in the joints, though not exactly like, either chemically or physiologically. It is disease of the heart caused by these deposits, and that caused by the deposition of fat in the substance of the heart and also that caused by atheromatous deposits, either alone or in company with cartilage and bone, with which we are most concerned.

14. With the pathological history of these diseases I shall have but little to do in this monogram. Their beginning, progress and cul-

mination, and the causes of their beginning, duration and termination, are pretty well known to observers, but just why cartilage, and bone, and fat, one or all, should be deposited slowly, so slowly as not to attract the attention of the victim, in the heart of a habitual drinker more than in the heart of an excessive feeder, we cannot explain; but the same thing is done in old age even in those who have lived the most regular lives. Therefore, for this reason alone it is probable, and we know by observation that it is certain, that excess in drinking induces premature old age, and the deposit in the heart of organic substances, analogous to the healthy tissues, but of a lower formative grade, composed of dwarfed or defective form elements, just as we observe in old age.

15. This slow, permanent unsoundness of the heart is caused by something equivalent to old age, and the result will be the same, death, unless the habit is changed and reparation made for loss sustained. *However much we do the result will always fall short of complete restoration of the heart to as sound a condition as it was before the deposit of the analogous tissues.*

16. We are accustomed to say that heart

disease comes of itself. If one has an inflamed eye, we say the eye has been recently hurt, or exposed to a cold wind or some contagion; if one has a fever, we say he has been exposed to its cause recently. We recognize these causes, and always refer to them for the solution of the phenomena of acute affections; not so with chronic diseases, we are apt to say they come of themselves; and yet they have causes as definite and appreciable as an intermittent fever or a pneumonia; but they are so long in acting that we keep no record of them, and do not seek them until the disease takes form and place.

We do not know why this premature old age seizes on the heart rather than the lungs or liver; why it does not at the same time cause wrinkles, and parchment coloration of the skin. These negative inquiries are unprofitable, and if answered would not elucidate the unsolved problem; why are analogous growths deposited in the heart at all? They are deposited because one dissipates. But why does dissipation cause them? Because it induces old age? But why does old age cause these deposits? Because nutrition is defective, and hence the form ele-

ments are dwarfed and imperfect. But why is nutrition defective in old age? Here the oracle returns no longer any satisfactory answer, and hence the first question remains unanswered and unanswerable; that is, we do not, and probably never can know why analagous growths are deposited in the heart at all.

CHAPTER III.

DIAGNOSTIC SIGNS OF ENDOCARDITIS PERICARDITIS, AND INSUFFICIENCY OF THE MITRAL VALVE. CASES. TREATMENT OF MITRAL INSUFFICIENCY.

1. We have seen that the first sound of the heart is caused by the closure of the auriculo-ventricular valves—mitral and tricuspid. *

2. That the second sound is caused by the closure of the semilunar valves of the aorta and pulmonary artery shutting back at the same instant.

3. Endocardial *murmurs* are distinguishable from exocardial *frictions*.

4. The ear cannot mistake a murmur for a friction, nor the contrary—hence when there is a persistent murmur the affection is surely within the heart.

* I do not venture to say that there is no other element in the causation of first sound, but the closure of auriculo-ventricular valves is the only cause of any practical importance.

5. When there is a friction sound, persistent or not, there is pericarditis acute or chronic; and the disease is outside the heart.

6. Now, if murmurs proceed from diseased valves, the murmur should, other things equal, be heard loudest when the ear is nearest the affected valve.

If the mitral valve is affected the murmur will coincide with a part or all of the first sound; and be heard most distinctly, either at the apex of the heart that is where the heart is seen and felt to strike the chest below the left nipple, or, a little above the left nipple, to the left of the sternum, between the fourth and fifth ribs, directly over the mitral valve. If the disease is of the mitral valve, the sound will be heard distinctly at the apex of the heart, and perhaps for some distance below it in the same right line; but if the aortic valves are affected, the murmur will be heard loudest just about where the mitral murmur was heard loudest; but in place of being heard more distinctly in the direction of the apex, it will be heard more distinctly in the direction of the aorta, that is over the sternum, opposite the second rib. (The aorta ascends behind the sternum, obliquely upwards and for-

wards towards the right side until it reaches the upper border of the second costal cartilage.) Whether the murmur proceed from the mitral valve or the aortic valves, the murmur will attend the systole of the heart; that is, it will coincide with the first sound. Whether the murmur proceed from the aortic valves or the mitral valve in nine cases out of ten it will be heard loudest in the precordial region, with the ear or stethoscope placed over the costal cartilage of the fourth rib. By listening over the precordial region only, no man can say which valve is diseased, because the stethoscope will cover all the valves if it is an inch and a half in diameter; and most physicians will confess that it would be impossible to say from which portion of the circle included in the mouth of the stethoscope the murmur proceeded in a given case, but if on moving the stethoscope towards the apex of the heart, the murmur should be heard louder than it would be if moved up the arch of the aorta, or in the direction of the pulmonary artery, the diseased valve would almost certainly be the mitral, if the murmur coincided with the hearts systole. In this case the murmur would result from the

blood being repelled backwards from the left ventricle into the left auricle, through the imperfect mitral valve. It will be borne in mind that in health of the heart the contraction of the left ventricle impels the blood forwards through the aorta. Now the aorta is composed of powerful elastic tissues, and hence it reacts with violence on the current forced through it by the ventricular systole, and it is obvious that when the aortic valves are open, as they are during the ventricular systole, that if the mitral valve were open also, there would be nothing to prevent the aortic resistence, together with the contraction of the ventricle from repelling the blood through the mitral orifice ; and it is also obvious that this repulsion would be synchronous with the ventricular systole, hence the resultant murmur would be synchronous with the same systole, and would be heard best, not in the direction of the aorta, but of the axis of the ventricle, that is in the direction of the apex of the heart, which is the apex of the left ventricle.

And by the same reasoning it can be shown, that if the murmur proceeds from the aortic valves, roughened with analogous growths, but

not so impaired as to prevent them from closing, that the murmur will be synchronous with the first sound, and heard louder in the direction of the aorta, that is of the onward current of the blood, than in the opposite direction, that is, towards the apex.

But again. If the mitral valve is sound, and a murmur be heard synchronous with the *second* sound, louder in the direction of the apex than in the direction of the aorta, or if it be about equal in both directions, there will be imperfect closure—insufficiency of the aortic valves and the murmur is not direct, but regurgitant, proceeding from the resilience of the aorta, repelling a portion of the current back through the aortic valves into the ventricle from whence it issued; but as this regurgitation is not absolutely synchronous with the forward current of arterial blood in the aorta, it would be expected that the murmur of regurgitation would be carried downwards rather than along the aorta, because the course of the blood at the moment of the development of the murmur is downwards into the left ventricle, in which direction the murmur should be propagated. But this is too nice a distinction to be always made

out definitely; for at times the murmur is so loud as to be heard distinctly in all directions from its origin, and in a radius of two inches no difference in pitch may be appreciable; but if the ear recedes some distance, it will always be found that in regurgitant aortic murmur the sound is propagated downwards, and is coincident with the *second* sound, while in merely roughened, but not insufficient, valves, the murmur is coincident with the *first* sound and is propagated upwards.

Diagnosis of Organic Disease of the Heart. 1.—First, it is premised that the auscultator or physician is aware of the following summary of facts, namely: that there are two sounds—a *first* and *second;* the *first* longer and less accented than the *second;* the *first* propagated downwards, in the direction of the heart; the *second*, upwards in the direction of the ascending aorta; the *first* coincident with the pulse, the *second* a moment subsequent to the pulse.

The *first* sound is caused by the closure of the auriculo-ventricular valves—tricuspid right side, mitral, left side of heart. (All the valves are situated under a little circle not exceeding an inch and three-quarters in diameter, which may

be described on the skin over the base of the heart.)

2. The *second* sound is caused by the closure of the semilunar valves of the aorta and of the pulmonary artery ; the valves of these two vessels close at the same instant, and hence, though each emit a sound in closing, the ear of the listener at the precordia appreciates but one sound. Now, if the two sounds are normal in pitch, and tone, and accent, and if the impulse of the heart is not excessively strong, nor remarkably weak, if no *friction sound*, or *bellows murmur* is heard, we say the heart is sound. But if a friction sound is heard over the heart, proceeding from the heart, there is organic disease of its outside.

If a bellows murmur, or rasping murmur, is heard over the heart proceeding from the heart, and the patient is not pale or anæmic, and if the murmur is persistent, there is organic disease of the inside of the heart.

a. A friction sound indicates pericarditis.

b. A blowing sound—murmur—indicates either acute or chronic disease of some portion of the heart's cavities.

c. A friction sound and a murmur at the same

time, indicate that endocardial and exocardial disease co-exist.

d. We never can be sure of pericarditis until we hear the friction murmur.

e. We never can be sure of endocarditis until we hear the blowing murmur.

3. Latham says, from simply listening to a murmur and asking no questions, we are not able to tell what it means. In one who a few days since, while in perfect health, was seized with fever, it means one thing; in one out of health for some time, with difficult breathing and palpitation, it means another; in one deformed from birth, another; and in the pale chlorotic girl, another. Yet it is the same kind of murmur, the murmur simulating the sound of bellows, in all; and being the same, it cannot entirely explain itself—it cannot be its own interpreter. It requires aid from concomitant circumstances to decide its meaning in each particular case. The aid it needs is often very little; but that little it must have, and then it tells its story clearly and explicitly.

Case 1*st*. Miss A. D., unmarried, aged twenty, pale and short-breathed, cold hands and feet, occasional flush on one cheek, sometimes a short dry cough, often dizziness, headache, constipa-

tion, palpitation, says she has been told by Dr. —— she has "heart disease" and is incurable. On listening at precordia, hear bellows murmur, with first sound loudest in direction of aorta; this murmur is persistent for a week, pulse 96, feeble, accented; heart sounds distinct and *snappish*, that is, unusually definite; eye-lids at times "puffy." She was assured she was anemic, and had no heart disease, and was curable.

Treatment.—To retire at 9 o'clock, to keep her feet dry, to eat beef-steaks and mutton-chops, and take three raw eggs daily, to drink coffee or chocolate, to work but little, to take passive exercise in the open air—(ride in cars or carriages).

The only medicine ordered, if medicine it could be called, was equal parts of cod liver oil, whisky and simple syrup in table-spoonful doses three times a day. Result: in two months the bellows murmur could be developed only after violent exercise, such as swinging dumb-bells. At the expiration of four months, both paleness, and murmur, and palpitation had disappeared. I should mention she had used vaginal abluents of a solution of tannic acid for leucorrhœa, which was thus arrested.

This was not a case of organic heart disease, proved by the crucial test of *complete* recovery.

Case 2d. Mrs. B., has been under my care eleven years, during which time she has had no acute disease, and during which time I have seen her, at least, twice every week. Bellows murmur, coincident with the first sound, heard loudest at the base and in the course of the aorta—swelling of the lower extremities so as to require shoes two numbers larger—little oppression of respiration, except on rapid fatigue, when the face becomes purple and congested, and the extremities pale ; frequent headache ; assimilation good ; tendency to corpulency ; appetite always good ; excretory functions well performed ; pulse eighty, not strong, split, irregular, intermittent from one to ten times per minute often when in repose ; much more frequently intermittent in sleep than when awake ; murmur audible, but the first sound is of the tricuspid ; *diagnosis*, thickening and roughening of aortic walls ; mitral insufficiency slight ; hypertrophy of left ventricle.

4. Theory of Diagnosis.—By exclusion ; the aortic valves shut back distinctly : murmur propagated upwards, not coincident with *second* sound ; hence aortic valves are competent ; no jugular pulse, hence tricuspid not incompetent.

By the thickened walls of aorta, left ventricle

is always overtasked ; hence hypertropied ; and from excessive work and the resistance of the thickened aorta, we have momentary forgetfulness of the heart (oublie du muscle), hence intermittent pulse and irregular pulse. The blood is impelled with diminished force, in consequence of inelastic aorta ; hence there is capillary engorgement and cellular infiltration.

5. Lastly : Theory of mitral incompetency : the murmur not heard loudest at apex ; were the murmur only mitral, it would be loudest at apex ; but it is a composite murmur, composed of element *a*, aortic murmur, element *b*, mitral murmur, the two absolutely synchronous, but *a* very much louder than *b*, so that by comparison total murmur is heard loudest in the direction of the aorta.

6. The quick exhaustion, the purple face after exercise, the almost daily headache, without or with constipation indifferently, the inability to endure close rooms or large assemblies, are so many affirmative signs of mitral disease. Add to this, the mitral murmur is heard with extreme distinctness as high as under the middle of left clavicle, and under angle of left scapula ; lower toned than over aorta, because it is a lower and different murmur. Increased second sound of pulmonary artery can not be made out positively.

Treatment.—Mrs. B. has taken about a quart of compound spirits of lavender, and four ounces of spirits of ammonia, about one ounce of quinine, about six hundred aloetic pills, has been bled at the arm twice, four to six ounces for angina pectoris with fever, has been dry-cupped and leeched at the precordia for pain in the heart during fever, has taken digitalis occasionally for excessive rapidity of heart-beats, has taken large quantities of bitartrate of potassa as a diuretic, for swelling of feet and legs, and this with a cathartic of oleum ricini has always afforded immediate relief to the circulation without impairing health. She has taken perhaps ounces of muriatic acid in three drop doses after eating, to promote digestion, which for the last three years has been painful without the acid. (The hint concerning muriatic acid in dyspepsia, occasioned by organic disease, I get from Trousseau.)

8. *Result.*—Her present condition is not unsatisfactory, when the incurable nature of the affection is considered. The hypertrophy has greatly increased; there is more frequent complaint of uneasiness about the heart; fatigue is not so well borne; digestion is accomplished with more difficulty; constipation is habitual; hematosis is less

complete ; the muscles are becoming flabby ; the extremities do not swell so much as formerly, because assimilation is less active, but on the whole she seems in pretty good health, and by treating symptoms as they arise, according to rational ideas now possessed by the profession, she may live yet many years ; perhaps ten, or even twenty, but without constant care and close observation, she might die in a few months, that is, simply by leaving the disease to take its own course. The object of treatment is, in this case, to diminish the labor the heart has to perform, by guarding the integrity of the functions of the skin, lungs, alimentary canal, renal organs, liver, etc., and last, but far from least of all, the nervous system, by preventing mental and moral disturbances, and keeping all causes of grief, depression, anxiety and hopeless desires out of the way. Late physiological researches have made it probable that no causes are so potent in hastening the fatal course of organic disease of the heart as those which act reflexly from impressions made on the nerves of special sense ; and, on the other hand, through these same means the advance of heart disease may be greatly retarded and occasionally held at a stand-still for months or years.

9. *Summary.*—Is there a certain infallible sign pathognomonic of incompetency of the mitral valves? I do not believe there is any *one* sign or symptom on which we can rely. But if there is a murmur heard loudest above the left nipple, synchronous with the heart's impulse against the ribs, and at the same time there is increase in the intensity of the second sound of the pulmonary artery, (heard in the second left intercostal space, about half an inch from the sternum, and propagated directly upwards), it is as certain as anything can be that there is mitral regurgitation. But this increased second pulmonic sound cannot always be made out; still we cannot say, therefore, that the diagnosis cannot be made ont. It cannot be made out with the same rigid certainty, but it can be made so clear as not to leave a reasonable doubt. For example: the murmur is heard above the nipple, the pulse alike weak in both wrists, patient not seeming in good condition as to blood-making power, uncomfortable feeling in the chest, quick exhaustion and lividity of face on rapid exercise, hypertrophy of heart.

10. With two or three of these signs well established, we are sure of mitral regurgitation; a persistently weak, irregular or intermittent pulse adds to the certainty of the affection.

a. All that is known of the cause of intermittent pulse may as well be suggested here, as there will be constant occasion of naming it, to aid in explaining organic lesions. The proximate cause of intermittent pulse is arrest of the systolic movement of the heart, and the remote cause of this arrest is a difficult and unsolved problem in pathology; but there is reason to believe its origin lies rather in a lesion of some portion of the cerebro-spinal axis, than in any alteration in the structure of the heart itself. This intermittent pulse is the clue that leads us to the remote cause of the organic alterations in the heart, viz.: a primary alteration of the molecular substance of those portions of the brain and cord which preside over nutrition and elimination. This is not an attempt at an explanation of the efficient cause of intermittent pulse, but a dim suggestion as to the places in which that cause is to be sought. It seems sometimes to depend on some obstruction acting like a valve at the orifice of the aorta. The ear is conscious of a systolic movement, and of its arrest when about to be completed; and during this momentary arrest there is neither sound nor murmur, but the blood itself seems to stand still in the whole arterial system; and, indeed, it no doubt does, for the arrest occurs directly after the diastole, and ex-

tends not only over the pause between that and the systole, but also occupies the period of part or all of the systolic movement; and sometimes the arrest is longer than a complete pulsation. In the latter case, there is a sense of suffocation, attended with feeling of profound exhaustion, and a feeling of "waves" in the brain, which latter feeling interrupts consciousness, and even sensation, for an appreciable instant. The patient often seems abstracted or pre-occupied, and gives postponed and undecided answers to simple questions, from inability to decide quickly and respond promptly, because the brain, from unequal supply of red blood is not always ready to command.

b. Intermittent pulse, considered by itself, is without significance in the present state of knowledge; but taken in connection with hypertrophy of the left ventricle, mitral incompetency and direct aortic murmur, it still adds to the dangerous import of other symptoms. It aids us in the discrimination of aneurism from valvular affection; for in the former the pulse may be absent from one wrist, but not intermittent in both; while in valvular disease nothing is more common than intermittent pulse. If there is aneurism and the pulse is intermittent, we may be sure there is valvular disease also; and it

will be quite probable that the mitral and aortic valves are incompetent, particularly the mitral. There is an intimate relation between the frequency of the heart's beats and intermittent pulse. Under the immediate effect of a dose of ammonia and lavender, the intermittence will disappear. It seldom or never occurs when the pulse is above 85. It is very common when the pulse sinks below 75, either from unknown causes, or under the influence of digitalis. Yet, if there be no organic disease, digitalis will not develope the intermissions ; but, however extensive the organic lesion, cardiac stimulants, during their action, abolish the intermission.

a. It is remarked above, that "it" (intermittence) "seldom or never occurs when the pulse is above 85." This needs explanation to seem exact. It is understood that the patient is in repose ; not under the influence of any mental, moral or bodily excitement, and that the heart is not driven beyond the limit of endurance, (*a*) by some temporary stimulus, for example : excessive indulgence in stimulating drinks ; (*b*) by violent exercise, such as running up hill, or with all possible speed ; (*c*) by fright or anger ; (*d*) by mania or delirium tremens ; (*e*) by the agony of *angina pectoris*, and the violent motions of desperation in the effort to breathe, such as beating the air,

clutching the throat, rapid change of position, etc., etc. Under any of these unusual circumstances or conditions, the pulses will "hang," or intermit, even while the ventricles succeed in accomplishing even 120 beats a minute, as in Case 3 below; but in such cases, when the usual condition of the patient, as to repose, etc., returns, the pulse will become slower; the intermissions will give place to simple irregularity in time and tone, and finally under remedies properly selected and graduated, the pulse will become regular in time first, and (if the dilatation of the heart is not too far advanced, or if the fatty degeneration be only incipient,) soon after in tone; but if the sedative—say digitalis—be continued until the pulse falls below 70, though it be full and strong, the intermittence will return, either as to a part of a systole, while the pulse in the intervals of the intermissions, say for ten, twenty, forty, or even a hundred beats, may be regular in time and tone.

b. When the heart is affected with organic disease, excessive increase of its function from external causes develope irregular, remittent and intermittent pulse; excessive retardation in the heart's beats induce remittent and intermittent, but not otherwise irregular pulse. It is not affirmed that similar causes never induce similar

states of the pulse in persons in good health, free from anemia, and free from organic disease; but one may venture to remark that such a result is at least improbable, and is rarely observed, though sought for with great diligence. The rhythm of the heart is so hedged about by all influences, that any material disturbance is almost inconceivable in the absence of alteration of the blood as to quality or quantity in special localities, (local congestions or local anemias,) or of abnormities of the heart, arteries, or cerebro spinal axis. But respectable gentlemen and respectable authors assert that intermittent pulse *per se* is no evidence of a pathological condition anywhere. One must be permitted to doubt the *essential* truth of unproved affirmations. Intermittent pulse is not a normal condition, and if persistent independently of volition, under certain definite conditions, such as those suggested above, it cannot fail to be of grave significance.

c. There are rare cases of rhythmical intermittence, depending on some congenital abnormity, and there are well attested cases, but entirely exceptional, in which the systoles are somewhat under the control of volition, and in which intermittence can be caused by mere volition; but such cases must be acknowledged to be ab-

normal, and they are so rare that they may be omitted in diagnosis.

d. The irregularity of the heart in anemia or spanæmia of any kind, is not inconceivable, on the theory here suggested.

e. The normal condition of the heart can be normally performed only when the organ is sound in structure, and supplied with proper nutrition in proper quantity, and when it has natural or healthy blood to exercise its function upon, and in quantity proportional to its dynamic structure. Depraved blood supplied to the corronary arteries may cause both irregularities and pain: even spasm, genuine *angina pectoris*, may be produced from the same cause. Anemic patients often complain of uneasiness and even positive pain about the heart, and I have seen recently a case of anemia in which there was fixed pain in the third left interspace close to sternum.

Precordial uneasiness in anemic subjects, is as constant a symptom as irregular and feeble pulse; and there is every reason to believe this pain and this uneasiness are the results of defective nutrition of the heart itself; and it is quite certain, as the anemic condition disappears, as red blood discs increase in number, the uneasiness and pain diminish and disappear at the same

time with the irregular pulse, and the complete restoration of the blood.

9. *Pain is probably always the result of abnormal or defective nutrition ;* and uneasiness is pain in a less accented degree.

h. Finally, extreme excitement and extreme depression develope intermittent pulse from the same cause—defective nutrition and innervation, either relative or absolute ; but in repose the pulse seldom or never intermits, unless it falls below 70 per minute, and then irregularity is less marked than remission or intermission.

11. In mitral regurgitation the heart increases more in width than length. It is important, when there is any doubt of the degree of semilunar incompetency attending the mitral defect, to take this fact into consideration ; for, if the heart is not sensibly elongated, the aortic valvular incompetency will be very slight or entirely absent.

12. *Treatment.—a.* For mitral regurgitation a great deal of passive exercise in the open air is of the first importance. This can be accomplished by riding, or sea voyages. Walking should not be recommended, nor indeed any considerable voluntary exercise ; but passive exercise cannot be too much or too constantly insisted upon.

b. Well ventilated sleeping-room—that is, a room in which air is admitted through an opening of two square feet, with no gauze or curtain over it; comfortable warmth to be maintained by bed-clothes, not by the exclusion of the atmosphere.

c. A quiet mind and an even temper aid greatly in prolonging life, and the prevention of suffering.

d. A meat is better than a vegetable or mixed diet; no objection to fruit; diet spare.

e. Arterial stimulants must be avoided.

f. Tobacco is very injurious in all kinds of heart disease.

g. Coffee is seldom tolerated with impunity.

h. If the pulse runs above 80, digitalis and solution of perchloride of iron are indicated.

l. If the pulse is very weak, *beef juice* must be given freely, say a pint a day.

k. If the patient is strong and full-blooded, a purge should be given whenever there is any tendency to congestions or effusions; the non-stimulating diuretics are always serviceable, given two or three days, at intervals of two or three days.

l. Frequent bathing is much and imperatively

required in almost all affections of the heart and arteries; plain water, hot, is quite as good as any medicated bath.

The labor of the heart must be lightened from time to time, in bad cases, by purging, by diuretics, and even by leeching and cupping over the base of the heart, or by leeches at the anus.

It is doubtful whether bleeding at the arm, or in any considerable quantity, would ever be indicated in mitral regurgitation. *The angina pectoris*, which comes on with such violent paroxysms, will yield to dry cups in two or three minutes, quite as certainly as to scarifications or leeches followed by cups.

13. *Case* 3. On the 12th of February last, Mr. ——— Turk street, with mitral disease and aortic incompetency, had a most frightful paroxsym. His face and hands were purple; he beat the air wildly with his arms: suffocation seemed imminent; his pulse had ceased; the heart beat 130 per minute; irregular and intermittent. He was bathed in a sweat of agony, gasping for breath, with doors and windows open. Four cups were placed over the region of the heart, but before the fourth one had been set, his paroxysm began to subside, and within less than five minutes, his pulse became tolerably

regular at the wrist, and the heart beats had fallen to 120, and much increased in strength. The cups were left on three hours, and, on being removed, contained more than a drachm each of serum and blood, which had been drawn through the skin.

14. I am not quite sure the cups would not do as well on some other part of the body; indeed, there is reason to believe they would, inasmuch as their beneficial effect must be principally reflex; but the patients always want something done about the region of the heart, where they say the anguish centres. *

15. I said above, *h*. "If the pulse run above 90, digitalis and solution of perchloride of iron are indicated." If there is bronchial irritation, with dark muco-sanguineous expectoration, I would add tartrate of antimony, in small doses, to the digitalis and iron.

a. The iron and antimony should be alternated with the digitalis, instead of being given at the same time. A drachm of the solution of the perchloride of iron with five grains of potassio tar-

* I shall have occasion in the following pages to allude to mitral disease many times, and will attempt to explain the mode of death in this affection in some subsequent paragraph.

trate of antimony in a pint mixture, composed of equal parts of distilled water and simple syrup, will constitute a preparation of which a teaspoonful or more may be given every two hours, with decided diuretic and tonic effect. If there is albuminuria, it modifies or arrests it very promptly, unless the kidneys, one or both, are acutely inflamed. If such is the case, leeches, or scarification and cups over the kidneys, followed by warm fomentations will be required before the diuretic effects of the iron can be realized.

b. The passage of the iron into the circulation and through the kidneys should be verified by adding gallic acid to the urine subsequently voided, when if iron be present, the urine will instantly become inky. I constantly employ this mixture of iron, or the mixture with the antimony omitted, as a tonic, diuretic, and a sedative to the heart; and it seems to me to greatly aid the action of digitalis. The perchloride of iron ad ministered thus largely diluted, is probably both preventive and curative of anemia, and thus it must tend to protect and restore the heart itself in the overwork to which it is constantly subject when organically affected.

c. It requires a strong will, an intelligent volition on the part of the patient to persist in a

proper and rational diet in affections of the heart. For this reason, it would seem fit that he should be early informed of the nature and tendency of his disease, and the reasons of the modifications which are requisite in diet, exercise, etc. It is a mistake to suppose a patient can be made materially worse by being informed of his true condition; the deterioration will be only temporary, and when the first fright or dread has passed off, the patient will be in a better condition for treatment than when oppressed with vague and irrational fears, or hopes which he will too soon learn are baseless or improbable. No medication will avail, without the most carefully and rationally regulated diet, both as to quantity and quality.

d. Nothing is more fatal than the theory of treatment which proposes absolute rest in bed, with low vegetable diet for organic diseases of the heart. It will hasten death in almost every case, by the additional injury it entails on the heart's substance, by deteriorating the quality of the blood supplied to the coronary arteries, and to the ganglia, which preside over cardiac innervation.

e. What we want is to diminish the whole weight of the body, blood and all, while the blood

is maintained at its normal standard of excellence, or as nearly so as possible with the constant progress of a disease whose tendency is always fatal.

f. I said (12 *e.*) arterial stimulants must be avoided ; this needs qualification. For example, the following :

Case 5.—The patient, now under treatment, has mitral regurgitation, with all its attendant and consequential ills; hypertrophy and dilatation of both ventricles, aortic semilunar, and tricuspid insufficiency, œdema of the lungs, and cough with venous blood-tinged mucous ; dropsy universal, orthopnœa uninterrupted, dyspnœa so great as to induce almost constant perspiration. *g.* Here are the clinical symptoms observed at last visit. A large-framed man, aged 35, is sitting almost perpendicular in bed, (his constant attitude,) his face is moist, his breathing labored, cough frĕquent, venous blood-tinged expectoration, universal dropsy, everywhere in lungs coarse moist rales, and occasional whistling respirations—no first sound at apex, but an indistinct murmur ; intensely increased second pulmonic sound heard over third left cartilage ; aortic second sound replaced by a murmur heard loudest on sternum, between ends of third ribs—

aortic regurgitant murmur—the radial pulse full, strong, visible, large, resisting and regular, and 120; the epigastric pulsation of abdominal aorta is much more forcible and visible than apex impulse; the carotids throb; when the two index fingers are placed on the subclavian arteries in the subclavian triangles, the arteries strike the finger ends at each systole, with the abrupt tap of a hammer—a short, quick, flat, decided blow. Mechanism of first sound appreciated by the ear at apex, but no sound; heart's impulse two inches to the left of nipple in fifth and sixth interspaces; systolic thrills, to the touch, all over the heart, especially in third and fifth interspaces, and third right space close to sternum. Jugular pulse; basic systolic murmur not audible above the second right cartilage; heart beats exceedingly tumultuous, with moderate but marked heaving impulse at the precordia; large, full veins everywhere on arms and legs; nails purple; a semi-livid hue to the whole body. Copious pink urates have been deposited for months; liver much enlarged; headache constant, but mind clear; appetite absent; digestion and assimilation very bad; bowels constipated; pulse 120; temporal arteries crawl like worms.

h. Now, I think there can be no question of

diagnosis in this case, nor of prognosis. Death is certain and imminent. In this case, though purgatives, diuretics, expectorants and even diaphoretics and perhaps precordial depletion are indicated, and absolutely necessary to the prolongation of life, even for a few days, yet stimulants, and tonics, and concentrated nutrients are equally and quite as imperatively required.

And this is the treatment pursued, and directly it is suspended, even for a few hours, all the symptoms and subjective sufferings are worse and much less tolerable.

i. Here whiskey, ammonia, especially the muriate, [because while it, the muriate of ammonia, unloads the liver and fortifies the heart indirectly, if not directly, it certainly diminishes the tumult of the heart, and steadies and retards its beats,] and perchloride of iron, and elixir of bark, and beef juice, and egg-nogg may be given not only with impunity, but with great advantage, and well-founded expectation of adding weeks, or even months, to life by their judicious use. Such a case should occupy a room twenty feet square and fifteen feet high, and the mercury should stand at 60, while two large windows should remain wide open day and night. He should also be wheeled about the room in a chair, that he

might have the benefit of passive exercise in the prolongation of life.

I am the more minute in all this detail of treatment because it is applicable to all forms of heart disease, with obvious restrictions and limitations.

CHAPTER IV.

DISEASE OF THE SEMILUNAR VALVES OF THE AORTA. DIRECT AND REGURGITANT MURMURS—DIFFERENTIAL DIAGNOSIS OF. TREATMENT OF DISEASE OF THE AORTIC VALVES, AND ITS COMPLICATIONS.

1. *The time and location at which the murmur is heard mark the lesion.* [Brown.]

2. A murmur with the heart's systole, louder in the direction of the aorta than in any other direction, indicates roughened semilunar valves of aorta, [provided that the chest is not deformed, and there is no anemia.]

3. A murmur coincident with the *second sound*, or replacing second sound, heard loudest between third and fourth ribs, left side, and close to the breast-bone, and hence downwards indicates aortic regurgitation.

4. The regurgitant aortic murmur is often heard distinctly down the sternum from the

fourth rib, and when so heard can only be confounded with tricuspid murmur, or the murmur of of aneurism of the descending aorta. It can be distinguished from the tricuspid murmur: *a*, because in the murmur from incompetency of the tricuspid there is jugular pulse coincident with heart's systole, while the aortic regurgitant is coincident with the diastole of the heart. *b*, in roughened but not incompetent tricuspid valves, the murmur is synchronous with the heart's diastole, that is with the second sound, and is propogated down the sternum; hence by auscultation and percussion alone, aortic regurgitation and direct tricuspid murmur cannot be distinguished; and it is probable that by the nature of the proposition these two affections can never be distinguished in this manner; but, if we hear the murmur loudest between third and fourth ribs, close to sternum on left side, coincident with the heart's diastole, or the *second sound*, (emitted by still healthy pulmonic valves,) it is certainly aortic regurgitation, and the murmur heard down the sternum will proceed from this cause alone, or from this and tricuspid roughness combined. But as the tricuspid roughness is of infinitely less consequence than aortic valvular incompetency, it may be merged in the latter and ignored in the diagnosis and the treatment, with-

out the least detriment to the patient.

c. We distinguish aortic regurgitant murmur from that of aneurism, by the fact that the murmur of aneurism is *always* synchronous with the heart's *systole*, while the regurgitant murmur is *always* synchronous with the *diastole*. If there is any difficulty in establishing these synchronisms, on account of rapidity of heart's movements, it can be remedied by giving small doses of digitalis, every half-hour, until the pulsations are reduced to sixty or seventy beats per minute, when the discrimination will not be very difficult.

25. There may or may not be dropsy of the extremities in aortic regurgitation ; and, if the feet and legs are swollen, this condition may be irregularly intermittent, depending upon the diet, activity of the alimentary canal, or of the functions of the skin and kidneys, by which the circulation may be so far relieved as to prevent or reduce cellular infiltration of the extremities.

26. All valvular diseases, in fact all diseases of the circulatory apparatus, are, *theoretically*, likely to produce dropsy of the extremities. But in practice, long standing cases of valvular incompetency [three years, Walshe] have been seen in which there was no œdema of the extremities.

a. If the capillary circulation depend upon the

vis a tergo, which consists in the ventricular systole, reinforced and supplemented by arterial elasticity, it is difficult to understand how if the *vis a tergo* is removed, as it is to a great extent. in incompetency of the aortic valves, there should not be uniformly hemic stasis in the capillaries, and consequential serous infiltration of the extremities. *b.* In point of fact, I believe that the cases of long standing in which dropsy of the extremities is absent are exceptional.

c. The following sentences of Walshe perhaps explain the frequent absence of œdema in this affection. "The cerebral capillaries can only be affected secondarily through the pulmonary class; *and the systemic capillaries are much in the same position.* Regurgitation may exist to the highest amount without œdema of the extremities." Grant that arterial pressure, aided by muscular contractions, valves in the veins, and diastole of the right ventricle, might keep the blood in equable and uniform motion through the capillaries, still how could the arterial pressure take effect and urge the blood towards the apex of a hollow cone, the base of which remains open, as it does in aortic incompetency? Until cases are cited to verify assertion to the contrary, it will still be probable *that the amount of œdema of the extremities will be one of*

the measures of the degree of incompetency of the aortic or tricuspid mitral valves. *

d. The sequential hypertrophy or that attending aortic incompetency, while at each ventricular systole it increases the distension of the aorta, and while this very distension increases the force of the succeeding recoil, must have a tendency, by adding to the impelling force of the blood, to diminish the tendency to capillary stasis and transudation ; hence hypertrophy in aortic regurgitation is alleviative until it becomes excessive, or until mitral incompetency sets in, and thus retrograde pressure through the pulmonic vessels on the right ventricle, and so on the cavas, and their venous and capillary tributaries, annuls the benefit of, or renders injurious the advancing hypertrophy.

e. When this stage is reached, the advance of hypertrophy is the advance of the whole disease towards a fatal termination.

27. Pain in the region of the heart is an exceptional symptom in either mitral or aortic valvular disease. There is always, if the patient's attention be called to it, a sense of discomfort, fullness, or pressure, or weight, or unnatural solidity about the heart ; and when aortic valvular and mitral diseases co-exist, as they do in the

* See Appendix, "Causes of Dropsy."

great majority of cases, the sense of discomfort is greatly increased, and there is generally considerable oppression of the chest, and often a dry cough, disposed to be paroxysmal, or to result from fatigue rather than to be continuous. To persons afflicted with aortic regurgitation, close rooms are intolerable ; any weight on the chest, or tight garments compressing the thorax, are unendurable, without great suffering ; violent exercise causes lividity of face and hands, and a sense of imminent suffocation. The patient often feels, on ascending a hill or stairs, as though he must inevitably fall on arriving at the top, and instinctively seizes something for support. There are occasional flashes of oblivion of all things, substantial or abstract ; a sort of suspension of every trace of intellection and special sense, for an appreciable instant ; there is reiterated tendency to *heart forgetfulness ;* that is, the heart seems inclined to rest, and is re-aroused by a sudden inspiration, or throwing up of the hands, or by a start.

28. The hypertropy of the heart that attends this disease is often very manifest to the naked eye, as well as to percussion. The ribs are seen to be sensibly higher over the left than right side ; the heart's impulse is felt over a wider space, and more to the left, and lower. There is

a metallic clang to the *systole*, and especially is the clang heard over the beginning of the pulmonary artery, *between the second and third ribs, left side, close to the sternum.* The precordial dullness on percussion is increased in extent, and in jecoral similarity. The *pulse* should be diminished in force, in consequence of regurgitation, pure and simple; but if the mitral incompetency, co-existing with that of the semilunar valves, is slight, and the hypertrophy considerable, the pulse may be strong and full, but will have an appreciable recoil, sufficient to suggest the idea of a backward current in the artery. In short:

b. The aortic semilunar valves are certainly incompetent *when there is a murmur without a sound, prolonged and heard above the base of the heart, synchronous with the heart's diastole; and when this same murmur is heard at the same time down the sternum, there being no jugular pulse, and when there is also hypertrophy and dilatation of the left ventricle.*

I must not omit the *sign pathognomonic* of Dr. Corrigan: that is, visible pulses of all the superficial arteries, and vermicular onward motion of the tortuous arteries; the artery moves forward in the direction of its axis—locomotion of the pulses—the radials, temporal and tibials, where

they run nearest the surface, are those in which the moving pulses are most frequently visible; "but in highly marked cases, the carotid, brachial, axillary, femoral and external iliac arteries distinctly present it." (Walshe.)

But although there are few or no cases of aortic incompetency without visible pulses, there are certainly many cases of visible pulses without aortic incompetency. I saw a case not many weeks since, in which all the superficial pulses were visible, a case of aortic aneurism and hypertrophy of left ventricle, in which the pulse was 120, when uncontrolled. The patient died of rupture of the aneurism into the esophagus; the aortic valves were perfectly competent, tested by water poured into the cut aorta, and by inspection of the valves. Though always present in, visible pulses are not pathogomonic of semilunar incompetency.

c. Hypertrophy of left ventricle is determined by percussion, locus of apex impulse, vigor of contraction.

d. Dilatation of ventricle is determined principally by clearness and quickness of the systolic movement of the heart; for though the *systolic sound*, first sound, be abolished by mitral and tricuspid incompetency, yet the systolic *movement*

remains as a determinable fact, and occupies an appreciable time, and takes place in an appreciable manner ; and it is by the manner and time occupied in this *systolic movement*, other things not opposing, that dilatation of the ventricle is made out. If all the facts mentioned in paragraph *b* are present, aortic valvular incompetency is absolute, (if there is no deformity in the chest of the patient, no tumor, etc..) but if one of those facts are wanting, the diagnosis lacks demonstration. For example, if hypertrophy and dilatation are absent, incompetency is improbable. If the murmur *down* the sternum be absent, the upper murmur may depend upon roughness on the inner surface of the aorta ; but in this case, the murmur will generally be *bounded by a sound*, that is, wil terminate in the click of closure of the aortic valves, though not always, especially if there is mitral regurgitation, fatty degeneration of heart tissue, constricted mitral orifice, or when the aortic valves or the aorta itself is thicker and less elastic than natural ; in all these cases, the sound, though its mechanism be present, may not reach the most practised ear ; but the diagnosis of aortic valvular incompetency is made certain only by the concurrence of the facts mentioned above.

29. As aortic incompetency and mitral incom-

petency are associate affections, the treatment of one is the treatment of the other, under limitations.

a. Aortic incompetency, in its beginning, may exist alone, or without mitral defect. In such case the lungs will not have become congested, as in mitral lesion, and we shall not have to contend with the annoying cough, or the spitting of dark blood, transuded from the pulmonary vessels. But there will be the same need of lessening the labor of the heart by all means consistent with the hygiene of the whole system. For example, in aortic incompetency, absolute quiet is indicated for that alone ; but absolute quiet is incompatible with digestion and nutrition, and the case would terminate fatally all the sooner in consequence of a low grade fever which would be generated. The treatment is rest, as to voluntary exercise, but passive exercise in the open air, without a day's interval, such as travelling by stage or rail, in ordinary vehicles, or on horseback ; the absence of all excitement and annoyance ; a light but nutritious diet. The patient may eat a little at a time, four times a day, much rather than an abundant meal twice a day. Small meals disturb the circulation much less than large.

But, unfortunately, few persons can follow the passive exercise treatment. They lack the means, or they are hindered by family ties, or the dread of being away from friends, or a disinclination to this species of exercise, and principally would they be hindered by a doubt of its efficiency: and this no doubt would increase, when after several months they found themselves no better; whereas. a patient should be abundantly satisfied, in almost all lesions of the heart, if he gets *no worse.* When we hold an affection of the heart in check, we accomplish all that can with any reason be asked of us. When we alleviate symptoms—*not entirely remove them*—we do all that a tolerably well educated patient will believe possible. We can prolong life, but we cannot say to our patient that he is no more liable to death than he would be were his heart perfectly sound.

d. In aortic incompetency, the patient is liable to sudden attacks of fainting, from anger or violent excitement, or over exercise. Absolute quiet, free exposure to the air, dry cups to the precordia, or if cups are not at hand, a few drops of boiling water over the base of the heart, or a mustard plaster will restore the circulation. A copious draught of cold water might cause instant death, by reflex shock to the already exhausted

heart. A spoonful of brandy and water, or a few drops of spirits of ammonia, with spirits of lavender and water, would aid immediate restoration ; hot applications to the soles of the feet, or leeches to the end of the alimentary canal, are slow but unobjectionable temporary remedies.

The bowels must never be constipated for a day ; this can be avoided by taking daily at bedtime an aloetic or compound rhubarb pill, or a pill of the following :

a. R. Aloës, Rhei, Comp. ext. Colocynthidis, aa, dr. ij ; Ext. Nucis Vomicæ, scruple j. Mix, and make, according to art, into sixty pills.

29. Formula *a* will be found convenient and efficient in almost all cases, and rarely contra-indicated. If dropsy should occur, either general or local, it is to be treated not as an idiopathic dropsy, *id est*, a hydremia, or anemia, but it must be met with purgatives, diuretics, low diet and diaphoretics, that the overloaded circulation which is its cause may be relieved as soon as possible ; and to assist this purpose, absolute rest should be enjoined, and the heart's action diminished with small doses of digitalis, often repeated, or digitalis alternated with aconite.

Digitalis has been objected to in many diseases of the heart by high authorities, (Corrigan, Hen-

derson, Walshe, etc.,) and especially in aortic incompetency. The argument against it is this, and nothing more :

a. Aortic incompetency admits reflux of blood into left ventricle, at the instant when the direct current is entering it through the mitral orifice ; hence the ventricle is overloaded and gradually enlarged—dilated and hypertrophied—the more frequent the heart beats the less the opportunity for regurgitation, and hence the less the ventricular engorgement ; but digitalis "slows" the heart beats, and hence allows more time for aortic regurgitation, and hence *increases* the dilatation of the ventricle.

b. Now, all this is true, but still it is no objection to the guarded use of digitalis. The argument against it deals with logical extremes, and is conclusive against digitalis from that point of view ; but the drug should be given to slow an *excited* heart, beating with *excessive* frequency, say more than 90 per minute, but not to reduce it below 80 or 85, for then it would become injurious, not only by favoring dilalation, but by increasing the already almost peculiar tendency to sudden death. Digitalis may be given, but with more caution than in any other valvular disease, and with quite as much caution as in

dilatation of the left ventricle. A minute dose often repeated—say every fifteen minutes—is infinitely safer than a larger dose at correspondingly long intervals ; a half a drop of the fluid extract every ten or fifteen minutes will often be found sufficient to keep the pulse at 85, when without it, it would ascend to 100 or 110.

c. I am in the habit of giving, empirically, in every affection of the heart, where there is uneasiness, pain or much discomfort, a fourth of a grain of quinine, with the sixteenth or thirty-second part of a grain of opium from three to six times a day. I double or quadruple the dose at times, when there is renal, pleuritic or hepatic pain, and patients more frequently ask for a repetition of this remedy than of any other ; but if there is headache, which is of frequent occurrence in valvular diseases, the smallest dose of opium appears to make it worse, while small doses, one fourth or one-half grain of quinine, often repeated, gives prompt and steady relief.

The weight of the patient must be reduced from time to time by diminished diet, such as lean meat and dried meats, with a little stale bread, in small quantities, without vegetables or liquid food. Even water should be drank sparingly. By these means the blood may be diminished in

quantity, *pari pasu*, with the decreasing weight of the body, and at the same time its quality remain unimpaired.

It is by no means desirable to add any form of spanæmia to organic disease of the heart ; and, although reduction of the heart's work is imperative, it is of the highest importance not to reduce its own strength and tone, while attempting to diminish the blood, which is at once it sustenance and its burden of toil.

The law applicable here is applicable in every organic disease of the heart, with modifications suitable to each case ; to wit : the heart must be aided by diminishing the labor it has to perform, without diminishing its own proper vigor or functional ability.

5. *Aortic obstruction*— constriction of aortic orifice—is indicated by a murmur coincident with the systole. The *first* and *second* sounds of the heart may remain unimpaired, but as aortic constriction must induce hypertrophy, we should find the impulse of the heart occupying a larger space, and the systolic shock increased, but in practice this is not always so. *

* If the aortic valves are also incompetent, the impulse will always be much increased.

But it may as well be stated here, that there is no known pathognomonic sign or signs of aortic constriction. Its existence may be made out with extreme probability, but never with the absolute certainty of mitral or aortic regurgitation.

6. Nothing can be determined by the pulse of exclusive significance ; it is likely to be regular in force and rhythm ; but if the hypertrophy of the ventricle is pronounced, it will be hard and and small, and have a wiry feel. Systemic dropsy is not a concomitant of aortic constriction ; cough is not present, although there must be tendency to engorgement of the lungs, when the blood flowing into the ventricle from the pulmonary veins, encounters, as it must, an amount of blood entirely excessive. There may be a slight cough, but if it is persistent, and the lungs not inflamed or tuberculous, and if there is no catarrh, either, constriction or incompetency at the mitral orifice must be suspected as a complication. A persistent aortic constriction entails (Walsh) a dilatation of the mitral orifice, and hence the murmur of mitral regurgitation from insufficiency, when the mitral valve itself is perfectly sound.

7. The anemic murmur is not persistent ; murmur from *unevenness of the internal surface of the ascending aorta*, is heard louder farther up the sternum than that from constriction of the orifice.

a. A marked fact in constriction of aortic orifice, is that the murmur is frequently heard in so many places and at such remote distances from its origin. It is heard under the right clavicle near the axilla; it is heard in the carotid, in the interscapular spaces, in all the intercostal spaces as low as the sixth, and in nearly as many of the left interspaces.

b. It is of slow progress towards a fatal termination, and is susceptible of becoming null as to its injurious systemic effects, by the hypertrophy of the ventricles, left and right. I say *left* and *right*, because this is the order in which their hypertrophy occurs in aortic constriction.

8. *Narrowing of the aorta* is sometimes a congenital condition, probably an intra-uterine arrest of development. The striking case mentioned by Latham exhibited the aorta and all its principal branches entirely free from disease, but more than one-half less than their natural capacity; the heart was found enormously enlarged and its cavities dilated. In Meckel's case (1750) quoted by Latham, the patient was a puny girl of 18. She had been from time to time subject to palpitation, and anguish, and trembling of the limbs, from infancy to her fourteenth year, and thenceforward the palpitation and anguish had

become constant and more severe until her death. Upon dissection, the heart was found enormously enlarged, and the aorta, throughout its whole course, especially through the chest and all its principal branches, marvellously narrowed. The heart had both its ventricles dilated, and their substance more soft than natural; its auricles also dilated, but the *left* to a degree far greater than any other cavity. It was capable of containing the enormous quantity of twelve ounces, while the corresponding ventricle only contained four. The aorta was not more than half the diameter of the pulmonary artery."

These were evidently congenital malformations of the aorta, and the enlargement of the heart was a necessary consequence, and perhaps an indispensable condition for the continuance of life.

But constriction of a short segment of the aorta will have the same effect on the heart as this general narrowing of the aortic trunk and its principal branches.

9. *The treatment* of aortic constriction is in no wise peculiar, and would be the same, with limitations, as that of aortic or mitral regurgitation. When the patient becomes over fatigued or excited, the pulse is very likely to become oppressively intermittent, perhaps to the extent of eight

or ten times a minute. When such a condition supervenes, absolute quiet should be enjoined, and the heart stimulated to regular contractions, by a dose of lavender and ammonia, or by dry cups. A little blood abstracted from the precordia by leeches, or scarifications and cups would almost instantly relieve the overtasked organ, not entirely by reflex action, but by absolutely diminishing the fullness of the vessels near the base of the heart, for it will be borne in mind that the internal mammary artery rests upon the costal cartilages, a short distance from the margin of the sternum; it sends branches to the upper part of the pericardium—*pericardiac arteries*—also to the skin covering the base of the heart—*anterior perforating arteries*—hence blood abstracted from the precordia would be abstracted from the near vicinity of the heart itself—namely, from the membrane investing it. And it is for this anatomical reason that local depletion is insisted upon in pericarditis by a late author (Markham).

But however this matter be explained, one thing is well known to all observers, that the local abstraction of blood in the vicinity of the heart's valves rapidly relieves all cases of *angina pectoris* in the various forms of unsound heart.

I am not oblivious of an apparent discrepancy in this statement with a fact previously stated, that neuralgic pain, of which angina pectoris is undoubtedly an example, depends on some local anemia. But this is more apparant than real—for the anemia may be relative or absolute—absolute when the total mass is diminished; relative when there is merely obstruction to its movement through some part. Again, a structure may be overwhelmed with venous blood in a state of stasis; for example, the arm below a ligature; the brain and upper extremities from a tumor pressing on the descending cava. In this case there would be pain in the head, perhaps, and yet there is excess of blood; but there is diminution of arterial blood circulating in the obstructed parts—this is relative anemia.

Excess of non-oxygenated blood is equivalent as to the production of pain, to diminution or defect of arterial blood—anemia. The pain induced by this venous stasis or retardation, might be relieved by local depletion, while pain from direct anemia of red blood should be relieved by arterial stimulants and hæmatogens. The pain of *angina pectoris* is probably often if not always caused by local retardation of the blood current, that is, by venous congestion—relative anemia.

But it cannot be ignored that this local depletion is much more applicable to acute inflammations of the inside or outside of the heart, especially of the latter, than to sudden accessions of congestion or anguish in chronic changes of the heart's structure, such as the affection under consideration—constriction of the aortic orifice. If the paroxysm or excessive palpitation in this chronic affection of the heart, or of most others of the same organ be not promptly relieved by dry cups, sinapisms, or by leeches or scarifications, the overtasked heart should be relieved by a small amount of blood taken from the arm. This is rational in theory and verified in practice. "I consider that I have seen life preserved by timely abstraction of blood (by venesection) in cases of chronic valvular diseases of the heart, where the organ was so overwhelmed and laboring as to render death imminent." (Markham.)

10. Care must be taken that too much blood, relatively to the condition of the patient, be not taken from the arm, lest by the alteration of the normal ratio of globules and fibrin, or by the feeble action induced in the heart, the formation of clot be favored, and death be caused in an instant. Such accidents have occurred, to the horror of the physician, who bled with the most

honest intentions, and under the influence of the dictation of the classic authors in medicine. But under the present rational ideas of the uses of blood-letting—merely to relieve an immediate and urgent necessity—there is little danger of a careful physician allowing more blood to flow than is absolutely requisite to relieve the congestion of the heart or lungs. Concerning the *treatment of aortic constriction* itself, we know absolutely nothing. All we can do is to prevent the accidental complications which attend it from suddenly destroying life.

11. Like all other chronic diseases of the heart, it has no known essential (denoting its essence) symptom ; and hence we cannot direct our treatment to the cure, or the arrest, or even the deterioration of the disease in its intimate nature. We see not the disease, but its effects ; namely, constriction, incompetency, etc. These are the anatomical alterations which the disease has produced, but they are not the disease itself. This lies behind the visible effect. It exists anterior to it in the blood, and in the ultimate structure of nerve and brain substance.

In chronic affections of this kind, then, we can only treat symptoms. But what are symptoms? Latham has eloquently and well said, " They are

not *mere* signs of the disease, but they are direct emanations from it; not things in themselves nugatory, but eminently real. They are natural sensations unduly exalted, or unduly depressed, or variously changed or perverted. They are natural functions hurt, hindered or abolished. So that a man may often with stricter propriety be said to be ill of his symptoms than to be ill of his disease, and, what is more, to die of his symptoms than to die of his disease.

Accordingly, it often happens, even where the disease is best understood, that we treat the symptoms of the disease only, just as if we had no knowledge of anything beyond them. Therefore, when we have confessedly no strict knowledge beyond them, (which is the case in chronic affections of the heart,) and the aim of our practice must need centre in the symptoms; we are not to lament over the shortcomings of our art, and its straitened capacity for doing good; for, it does not follow that, if we knew *the disease* ever so well, we would treat it otherwise than we are now treating its symptoms, or that what we are now doing for the symptoms would not be the best and would not be all that could be done for the disease itself."

Aortic regurgitation cannot be mistaken for aor-

tic constriction or obstruction. In the former case, the murmur is coincident with *second* sound—of the pulmonary artery, when that of the aorta is replaced by the murmur—in the latter case, the murmur is synchronous with the first sound, and both *first* and *second* sounds are audible. It is more common in old people, but young people are by no means exempt from it.

Were I called upon for an opinion in a suspected case of this kind, I should attempt a diagnosis by exclusion. It could be determined that the murmur was not mitral regurgitation from its location and direction, and from the persistence of mitral *first* sound, if the mitral orifice were not also incompetent. It is not aneurismal from the absence of dysphagia, of sense of a foreign body in the thorax, of thrill on digital pressure in the intercostal spaces. It is not aortic regurgitant, for it is not coincident with the diastole, etc., etc.

I must not leave the consideration of alterations of the aortic orifice, without alluding to an unexplained fact in connection with aortic regurgitation; that is, extreme tendency to instantaneous death.

The patient drops dead without the slightest premonition, and probably without any excita

tion of the circulation, as well as when walking or when motionally excited. The freer the heart is from any other lesion, Walshe says, the more likely is the patient to instant death from aortic regurgitation. The assurance of life is much longer if to the aortic, mitral regurgitation and ventricular hypertrophy be added. Even the addition of that most fatal of all valvular affections *tricuspid insufficiency*, is preferable to aortic regurgitation, pure and simple.

The instant death can be guarded against, theoretically, by seeing that blood be not made in excess, in fact that it be abstracted from the region of the heart, from time to time, if there is the least evidence of labored circulation.

Death occurs from syncope. It is a "fainting away" from which the patient never awakes. This peculiar tendency in aortic regurgitation was first publicly noticed by Chomel, and next by Aran. (Walshe.)

I have condensed the following additional observations on this subject from the *Gazette des Hopitaux*, 9th June, 1860.

"In 1844, M. H., Minister of Finance under the reign of Louis Phillipe, was working one morning with one of the ministry. The absence of a document compelled the latter to absent himself for

a few moments. When he returned, five minutes or more after his departure, he found the minister with his body inclined backwards over his chair, his right arm hanging over the arm of the chair, still holding the pen with which he had designed to write his signature to the document. All the means employed in such cases were resorted to in order to restore M. H. to life, but in vain; he was dead.

The autopsy, made by Blandin, revealed no lesion either in the brain or lungs. The heart was very large and loaded with fat; its right cavities, distended with black blood, were a little dilated and very much thinned. The left ventricle was greatly hypertropied and enormously dilated. Excepting a few nodosities, the mitral valve was healthy; but the aortic orifice was much constricted, and the sigmoid valves entirely ossified. There were a few cretacious deposits in the aorta.

It is important to add that the health of M. H. had exhibited no grave derangement which could have presaged the sudden stroke which terminated his existence. He had never had any of the general symptoms pertaining to diseases of the heart.

"Thirteen years after, in 1857, Mauriac, fre-

quently saw at *La Pitie*, a sub-officer of the guard of Paris, who had long frequented the hospital for advice concerning palpitation of the heart, and a constant difficulty of respiration. In this case there was ascertained to be a double *bruit de souffle* at the base of the heart, and the diagnosis was aortic insufficiency. He had neither dropsy nor anasarca. For many months M. Mauriac did not see this patient, when he learned that he had died suddenly. One of his comrades, who was on duty with him, left him alone to copy some document; when he returned, at the expiration of a few moments, he found his friend dead in front of his desk. At the autopsy, no lesion was found anywhere, except insufficiency of the valves of the aorta, with hypertrophy and dilatation of the left ventricle of the heart.

One year after this, Mauriac observed the following case: A young man, twenty-six years old, of robust constitution, about five months before entering into the hospital, had suffered for a fortnight with pains under the left false ribs. Six weeks before, he had palpitation for the first time, and one month previous, that is, on the 3d of March, he was taken with obstructed or impeded respiration and pains in the left groin, which radiated along the lower extremity of the same side. The 2d of April, his condition was

as follows : Slight emaciation : partial distension of the veins of the neck ; visible arterial pulsations ; skin warm and moist ; 88 to 92 pulsations, vibrating, irregular, intermittent at the arterial diastole from time to time. The heart is considerably enlarged. The maximum of impulsion takes place in the fifth intercostal space, 11 centimeters * from the axis of the sternum ; it measures vertically 11 centimeters upon the median line, and more than 16 centimeters obliquely from right to left. The impulsion is thrilling, and accelerated at times, as if the heart struggled to overcome an obstacle. The ear applied over the apex, perceives a double *bruit de souffle*, the second much softer, more mellow and prolonged than the first. Ascending towards the base of the heart, this double stethoscopic phenomenon persists and seems to reach its maximum of intensity under the second and third intercostal spaces, on the left side, in the vicinity of the sternum, also under the corresponding portion of this bone. This double bellows-murmur is also very marked in the aorta, the dullness of which is sensibly increased. Finally, in whatever place the ear is applied, there is not heard

* A centimeter is four-tenths of an inch nearly ; a millimeter is .039 inch.

any trace of the second normal beat of the heart. Bellows-murmur diastolic, intermittent, and very strong in the carotids. There is neither fever nor œdematous swelling of the abdominal extremities.

The 8th of April, the 6th day after his entrance into the hospital, the patient died suddenly, at seven o'clock in the morning. During the last few days of his life he complained of a feeling of severe oppression and a sensation of uneasiness at the epigastrum; the morning of the day before his death, he complained of great difficulty of respiration; however, he still kept about; in the evening he supped as usual; during the night he had some suffocating attacks, his sleep was agitated and painful; at seven o'clock in the morning he awoke and lay tranquilly in bed. All at once he lost all consciousness, his head fell back, his limbs straightened and were convulsively agitated, his face became rapidly purple, and almost immediately grew pale, and in less than ten minutes he died.

The heart, emptied of its clots, measures 0.13 centimeters from base to point, also transversely at the level of the base. The pulmonary orifice was 0.075 millimeters in circumference. The ventricle is a little dilated and hypertropied.

The right auriculo-ventricular orifice is somewhat enlarged ; its valves are supple and transparent ; the left presents some granulations on the auricular surface. Very marked dilatation of the right auricle.

The aorta is dilated immediately after its issue from the heart in such a manner as to assume a position more anterior than usual, and crowd the pulmonary artery backwards; the dilation extends as high as the origin of the left subclavian ; it has, however, preserved the flexibility of its walls ; finally, in the neighborhood of its orifice it exhibits a beginning of atheromatous alteration. The left ventricle is enormously dilated. The dilatation affects solely the arterial portion, and the mitral valve is forcibly pushed backwards, also the fleshy columns inserted into it, which latter are atrophied rather than hypertropied. The diameter of this ventricle is 7 centimeters, the thickness of its walls 13 millimeters. The mitral valve is supple, but the tendons which subtend it are thickened. Insufficiency of the sigmoid valves of the orifice of the aorta. A clot is prolonged to a height of 52 millimeters above the origin of the vessel, and adheres to the greatly altered right valve. The anterior and posterior sigmoid valves are thick-

ened and indurated, and have lost their elasticity, and contain, particularly at their bases, cretaceous and cartilaginous granules; their separating partition is much thickened. The right valve is almost entirely destroyed at its middle.

Dr. Mauriac proceeds to investigate the mechanism of sudden death, to study the elements of prognosis, and the therapeutical indications to be deduced from a knowledge of this mechanism.

When there exists, says M. Mauriac, an insufficiency of the sigmoid valves of the aorta, and when the hiatus which is the consequence of this is insufficiently large to permit the re-entrance of a considerable quantity of blood, at the instant of the heart's diastole, into the interior of the left ventricle, the latter becomes more or less distended, and it is compelled to increase its activity to impel into the whole arterial system this excess of blood, added to the amount which comes from the lungs. There results a hypertrophy of the walls and a dilatation of the cavity of this ventricle.

So long as these lesions do not go beyond a certain point, and particularly so long as they remain simple, that is to say, not complicated with any other morbid change, they oppose the stagnation of the blood in the lungs and in the

cardiac cavities; and are, therefore, salutary because they prevent the insufficiency from producing any considerable perturbation in the equilibrium of the general and pulmonary circulation. But hypertrophy and dilatation of the left ventricle of the heart, consecutive to valvular insufficiency, tend almost always to indefinite increase; and in proportion as they increase they lose more and more the character of a, so to speak, normal and physiological hypertrophy and dilatation, because they become the seat of secondary lesions, which embarrasses the circulation in the heart's own vessels and alter its muscular fibre.

Nevertheless, when the hiatus of insufficiency is small, when there is not considerable stricture of the aortic orifice, or complete rigidity of the aortic valves; when the inflammatory morbid process which has produced the insufficiency and the lesions at the origin of the aorta, is arrested and definitively extinguished, it is possible that the hypertrophy of the left ventricle having reached that degree which the maintenance of the equilibrium of the circulation requires, will desist in its progressive development and remain stationary during the remainder of life. M. Mauriac considers it certain that such a condition exists in those individuals who have been for a long time affected with aortic insufficiency, and

who finally suffer only slight inconveniences referable to the heart, such as palpitations or momentary dyspnœa, etc. He has seen cases in which auscultation left no doubt of the existence of an insufficiency of the aortic semilunar valves, who had not even supposed they had any disease of the heart ; percussion in these cases revealed only slight enlargement of the heart. The hypertrophic process had no doubt subsided, and to this cause they were indebted for their undisturbed health.

Unfortunately, hypertrophy and dilatation of the left ventricle rarely remains circumscribed within these limits. There are many causes which tend constantly to augment these two lesions in which consists all the danger of insufficiency, and the action of which culminates in perturbations of the heart's own circulation.

The organo-pathological development of hypertrophy and dilatation of the left ventricle, consecutive to sigmoid insufficiency, presents three stages : one stage of simple and salutary hypertrophy and dilation which augments the labor of the heart, and may counteract in part for the injurious effects of the valvular alterations ; a stage of hypertrophy and dilatation complicated with the preceding lesions, and with more or less fatty

degenerescence. Fatty degenerescence is very frequent in aortic insufficiency; it is the last step of morbid organization of which the ventricular walls become the seat, when hypertrophy and dilatation are not arrested in their first stage. Among the numerous causes of this complication, M. Mauriac thinks that which plays the greatest part is the progressive enfeeblement of the interstitial circulation of the heart, either when this enfeeblement depends upon aortic insufficiency itself, or, at the same time, upon alterations in the origin of the aorta and cardiac arteries. Still, as these alterations run a course essentially chronic, as well as the valvular lesions, it results that it is only at a very advanced period of cardiac disease that degenerescence is observed.

When it has invaded the whole organ, there is observed a considerable thinning of the walls, dilatation and extreme flaccidity. Hypertrophy of the walls is, then, in this phase of the malady replaced by their atrophy; but the dilatation persists and tends even to become increased, if death does not intervene and suddenly remove the patient.

Hence, we arrive at the conclusion that *the organic cause which induces sudden death in cases of aortic insufficiency, is hypertrophy and dilatation of*

the left ventricle, complicated with congestion or degenerescence of the heart.

From the instant these lesions have taken place, the patient is momentarily in danger of sudden death. Finally, this mode of the termination of life never occurs in those cases that, consecutively to innoclusion of the aortic valves, have only moderate dilatation and hypertrophy. All the observations collected by M. Mauriac prove this fact in a categoric manner.

These premises admitted, he explains the mechanism of sudden death.

The heart, the centre of sympathy, upon which are accumulated, by reflex movement, all the impressions of the organism, whether morbid or physiological, becomes, in consequence of the lesions suffered, excessively susceptible to the perception of these impressions. Thus, every moral perturbation, every affection of the mind, which disturbs or tires the centres of innervation, all fatigue proceeding from excessive muscular exercise, or from whatever expense of nervous influx, are so many causes which may accidentally induce death.

Under the influences of any of these causes, the ventricular systole is enfeebled, becomes incomplete, drives into the arterial system only a por-

tion of the blood which was accumulated in the ventricular cavity, at the moment of the diastole. But the elasticity and contractility of the aorta and its branches not being diminished, it re-acts upon the blood-columns, and impels a recurrent jet through the hiatus of insufficiency with the same energy as previously.

It follows that, after the second systole, the heart is overcharged with a mass of blood exceeding the amount it contained at the instant it was surprised with the debilitating action of the accidental cause. If it recover its energy, the circulating equilibrium may be restored ; but if its feebleness continue, the difficulty increases, and the quantity of blood which accumulates in its cavity is augmented at each systole, and at each reaction of the aorta upon the liquid column which is no longer shut off from the heart by the complete partition which is made in the normal state by the aortic valves.

The accumulation in the ventricular cavity of the blood which comes from the lungs and the different arteries, dilates the left ventricle more and more ; and, finally, it can no more contract.

From all that precedes, it follows, that as to the prognosis, that of all the diseases of the heart, there is none the result of which is so difficult to

be foreseen, or that exposes the physician to more deception and risk of mistake.

Here are a few signs which may enlighten him upon the gravity and result of the affection:

Shock of the point of the heart several times the thickness of a finger outside of a line vertical with the left nipple, energy of its impulse, absence of normal resonance over a considerable extent, indicating that the hypertrophy and dilatation of the left ventricle have reached that degree where sudden death by instant cessation of the movements of the heart becomes imminent; all these are signs of the worst significance.

In general, only uncertain inferences as to the result can be drawn from abnormal murmurs.

The *depressibility* of the pulse, which is observed at its highest degree when the hiatus of insufficiency is very large, should be regarded as a sign of bad import. Among functional troubles, accessions of dyspnœa, above all, when accompanied with violent palpitations, are also of serious significance; it is the same with syncope, which, extremely grave in all affections of the heart, without exception, is much more so in aortic insufficiency than in any other disease of the organ.

The resources of science are, unfortunately, too often powerless in this affection. All the efforts of the physician should be directed against the most imminent danger, that of syncope, by all the hygienic means susceptible of detaining in its progress the evolution of the organic lesions. The facts demonstrate the danger of the method of Valsalva, of which indications have been imagined to exist in the fullness and apparent force of the arterial diastoles. Bleeding should be practiced only with very great moderation, and only in cases where there exists an engorgement of the lungs which impedes the general circulation.

Sedatives and *cardiaco-vascular hyposthenisants* are indicated as palliatives in all cases where there are violent palpitations, dyspnœa, or precordial anxiety. Finally, the true curative indications are deduced from the course of the morbid process, of which the walls of the heart are the seat; it is especially by revulsive treatment, (cupping, scarifications, temporary blisters, cauteries, etc.,) that the indications of treatment are fulfilled.

CHAPTER V.

VALVES OF THE PULMONARY ARTERY. CONSTRICTION OF THE MITRAL ORIFICE. CONSTRICTION OF THE RIGHT AURICULO-VENTRICULAR ORIFICE.

1. Skoda says he has never observed *deficiency of the valves of the pulmonary artery, or constriction of its orifice, in consequence of faulty valves.* [By "faulty" he means diseased or altered, but not defective or incompetent.] Should incompetent pulmonic valves occur, the incompetency would be certainly recognized by a murmur in place of the second sound, of the pulmonary artery, and this murmur would be heard loudest between second and third ribs, near sternum left side, and from that point directly downwards. There would be hypertrophy and dilatation of the left ventricle in a less marked degree than in aortic regurgitation, and hence the impulse of the heart would be felt over a less extent than in the latter affection. The murmur of pulmonic regurgitation has a sighing quality, (Hope) not observed in aortic regurgitation. This sighing murmur is also heard in connection with diminished *second* pulmonic sound, when the valves of the pulmonary artery are slightly defective, but not so much so as to prevent the flapping of the valves. (Hope's experiment.)

2. The pulmonic valves are also liable to become roughened, in a chronic manner, from exudation of plastic matter, or from the deposition of analogous tissue, without destroying their competency. In such case there would be a murmur synchronous with the systole, heard loudest in second left interspace, and propagated directly upwards.

3. Hope never saw or found recorded a case of pulmonic regurgitation from disease of the pulmonary valves. He created the murmur artificially in the heart of an ass poisoned with woorara.

4. The slightest compression, as of a tumor, a congested portion of lung, a foreign body upon the pulmonic artery or aorta, produces murmur and thrill synchronous with the heart's systole.

5. The *second* sound, much diminished, might be heard in many places on the chest, even were the pulmonic valves utterly incompetent, if those of the aorta were still sound ; but the *second* sound heard would proceed from the *aortic*, not the pulmonary valves ; and so, when the aortic valves are incompetent, so as to furnish no sound, a much diminished second sound will quite probably be heard in the second left interspace, proceeding from the sound pulmonic valves ; for the

second sound is caused by the *shutting back*, at the same instant, of both sets of sigmoid valves, just as the *first* sound results from the synchronous closing of right and left auriculo-ventricular valves. *Practically, lesions of the pulmonic valves may be ignored in the diagnosis of unsound heart.*

6. *Constriction of the mitral orifice.* Diastolic murmur from this cause is exceedingly rare, even when the constriction is considerable ; this defect of murmur results from the feeble force with which the blood flows from the left auricle into the left ventricle. The auricle is a feeble muscle, and not being supported by a valve, could hardly inject the blood flowing into it from the pulmonary veins into the ventricle, with force sufficient to develope a murmur, unless the constriction should not have a *very* small orifice, and even then the murmur must be feeble, and would certainly be preceded by a loud murmur of mitral regurgitation.

7. Mitral diastolic murmur is certainly very rare. Latham says it is a clinical curiosity ; there is no murmur because there is no impelling force from behind. Markham met with less than a dozen cases in eight or nine years. *a.* Austin Flint, on the contrary, seems to have been peculiarly fortunate in detecting this murmur ; he

says: "It [mitral direct murmur] is by no means so rare as has been supposed. I saw at one time last winter six cases of it in Belle Vue Hospital, and several others at Blackwell Island Hospital."

b. I have never yet been able to detect mitral direct murmur, although I have had many cases in which there could scarcely be a doubt of mitral constriction.

8. Sometimes the murmur is attended with a palpable thrill at the left apex—*fremissement cataire;* in well marked cases where the action is vigorous, a loud and prolonged murmur is heard at apex and upwards, in direction of left nipple. This affection causes hypertrophy of *left* auricle and *right* ventricle, pulmonary congestions; aortic sounds and beat of arteries, weak in proportion to constriction; hæmoptysis of venous blood frequent and easily excited; breathlessness easily induced by over-exertion; rest and quiet in an especial manner produce ease and comfort. Skoda's symptom, increased second sound of pulmonary artery, is here well marked, and relatively intensified on account of the weakness of the *left* ventricular systole and diminished second sound of aortic valves.

9. WALSHE has never heard this murmur of great intensity, nor high in pitch; it is, however,

sometimes prolonged. The murmur is sometimes wanting, where the constriction is found after death. The absence of the murmur may be reasonably attributed to weakness of auricular systole, or to smoothness of constricted orifice. The murmur comes and goes even when orifice is greatly contracted: this is probably due to varying force of heart's action.

10. SKODA says, in mitral constriction hypertrophy occurs rapidly; that it is attended with a prolonged murmur, extending even into the period of the systole of the ventricle. During systole no sound or an indeterminate sound, in heard in left ventricle; again, a murmur (systolic) may he heard there, because deficiency of the mitral valves is generally associated with constriction of mitral orifice. Sounds of aorta weak, heart's impulse increased and perceptible even, over a greater extent than natural.

11. HOPE says the murmur is feeble, soft, usually on a lower key than a whispered *who;* absent unless the contraction is considerable; absent also when the contraction is *great*, providing the current is perternaturally weakened by softening, or extreme dilatation of heart, or by both. In such cases, there is almost invariably murmur of regurgitation; hence the disease of the valves

would not be overlooked. This murmur is exceedingly rare, originally thought frequent by confounding it with a murmur of regurgitation. "I have never known purring tremor accompany a diastolic mitral murmur, the current being too feeble to produce it." When contraction is great, pulse is more or less small, weak, intermittent, irregular and unequal, in consequence of the supply of blood to left ventricle being insufficient and irregular. C. J. B. Williams, quoted by Hope, says: "When there is contraction of the mitral orifice, there is usually more hypertrophy than dilatation in the left ventricle."

12. In this last quotation, there must be perfect accord among all thinkers. Also in the dilatation with hypertrophy of the left auricle and right ventricle, but the discrepancies concerning the *thrill—fremissement cataire*— from mitral disease are seemingly irreconcilable. Markham says, it is frequent. Corvisart says, it is "*sensible à la main appliquie sur la region precordiale.*" Hamernik says, it is like the hum of a spinning-wheel. Skoda says that in certain cases vibrations are felt when the hand is laid on the precordial region—the *fremissement cataire* of Laennec.

13. Hope never heard or felt it, and does not be-

lieve it occurs as a consequence of mitral obstruction. Walshe has never observed the thrill, but believes it possible.

14. There is a case in this city now in which the thrill is indubitable; but it no more resembles the purr of a cat or the hum of a wheel than it does the filing of a saw—but it does accurately simulate the *thrill* felt by placing one's finger on a gum-elastic tube, a few lines in diameter, connected with a hydrant with the water turned on. The obstruction of the water produces a palpable thrill and an audible murmur closely simulating mitral murmur and thrill; but in the case to which allusion is here made, mitral regurgitation is undoubted, yet neither Walshe nor Hope ever observed the thrill in any valvular disease. It is probable that the occurrence of the *thrill* is extremely rare, and in a diagnostic point of view entirely valueless, for pressure on any blood-vessel by any substance might develope a *thrill* under favorable circumstances.

15. I cannot leave this disputed ground without one more allusion to Skoda's very positive dicta —yet it should be observed that his assertions are unsupported by accompanying clinical facts, and that while they seem logical, they are nullified by the case cited by Walshe, and by two or

three others cited by Hope, where mitral constriction was *great*, and yet there was no diastolic murmur.

16. But to Skoda; he says: "The more constricted the mitral orifice is, the longer will be the time necessary for the flow of the blood into the ventricle, and the more prolonged and louder the murmur. It is in cases of this kind especially that vibrations are felt, when the hand is laid upon the præcordial region,"—the cat-purr of Laennec.

17. The first clause of this quotation is obviously exact; but the second, "the more prolonged and louder the murmer," is doubtful in theory, and contradicted by the facts above alluded to. The blood flowing from the auricle through a *smooth, circular or regularly eliptiform* contraction into the ventricular cavity, would be in the last degree *unlikely* to develope a murmur, however great the hypertrophy of the auricle. Liquid flowing through the constricted *end* of a tube, if the constriction be *smooth*, does *not* generate a murmur, unless the impelling force be relatively (to the size of the stream) very great.

That is, murmur depends not on the contraction or roughness merely, but on the momentum of the liquid flowing through the aperture; and

in mitral constriction the momentum will be slight, except in cases of great hypertrophy of left auricle, and only in such cases will murmur be likely to be developed, and then it will occur only at the moment of the auricular contraction, that is, barely pre systolic—so near the systole as to be easily confounded with regurgitant diastolic murmur; and as the mechanism of this murmur is the same as that of the regurgitant, it should be no mere sighing, but should have the same abrupt blowing character. Liquid flowing with great velocity and continuously from the end of a tube emits a sighing murmur, but not when it depends on an interrupted *vis-a-tergo*, like the contraction of an auricle suddenly. But liquid flowing through a constriction in the continuity of a tube, whether the constriction be rough or smooth, will be attended with *murmur*, and if rough with *thrill* also; and in cases where the thrill is manifest, it would be well to exclude *aortic regurgitation*, and even mitral *regurgitation*, by the most rigid logic, before attributing the *thrill* to *constriction* of the mitral orifice.

18. But there being *necessarily* any murmur at all, and especially its being attended with *palpable or visible* thrill would seem to be as much opposed to theory as to facts. Still, it would be extremely

fortunate if we had some sign pathognomonic of mitral constriction, for it is the one of the affections of the heart next to aortic regurgitation and tricuspid constriction most likely to terminate rapidly and suddenly, and it is one, if recognized positively, that may be treated with every expectation of prolonging the life of the patient far beyond the limit it would reach if the case were left to itself. Verification of hypertophous dilatation of the left ventricle, exclusion of aortic regurgitation, the presence of pulmonic congestion and hemoptysis, increased second sound of pulmonary artery, a feeble, irregular, unequal, intermittent pulse, would render positive the existence of mitral constriction, even if no murmur at all were present.

19. Thus in constriction of this orifice, if a murmur be developed at all, it will immediately precede a systolic regurgitant murmur, and will be propagated towards the left apex, and diminish and disappear before arriving at the second interspace.

20. Constriction of the mitral orifice would add to the unfavorable prognosis of mitral regurgitation. The same principles of treatment are applicable here as in other cardiac lesions. The heart must not be allowed to beat too fast—more

than 80—the weight of the body must be diminished, while the quality of the blood is maintained at a healthy standard. Abundant passive exercise in the open air, more a meat than vegetable diet, a quiet mind, slow bodily movements, baths, diuretics, aperients, occasional precordial counter-irritation, or even depletion; all these may be requisite, according to the condition of the body and the general health. It is not possible to follow any prescribed details of treatment for any length of time. The treatment must be constantly modified, to suit the ever varying exigencies of each case.

CHAPTER VI.

INORGANIC MURMURS—HOW DISTINGUISHABLE FROM ORGANIC.—DIASTOLIC INORGANIC MURMUR.

21. In a former chapter, two diagnostic signs, one of *endocarditis—a murmur;* one of *pericarditis—a friction* sound, were given. It was not said said that the affections could exist without these relative diagnostic signs; but it is not probable that an endocarditis ever occurs and runs its course without developing and maintaining during much of its progress a bellows *murmur*. It is not probable that the friction sound is ever absent at all times during the course of a pericarditis.

a. The *friction* sound is less persistent in pericarditis than murmur in endocarditis, because of the early effusion of serum in the former affection by which the serous surfaces are kept apart, and thus friction prevented. But as no such effusion takes place in inflammation of the heart's cavities, the *murmur* indicating it is likely to be persistent until its termination in death, recovery, or in permanent unsoundness of the heart without inflammation; in the latter contingency, the *murmur* continues and becomes an index of chronic disease of the valves.

22. But murmurs may exist and closely simulate endocardial murmur, from inflammation of the *inside* of the heart, or from valvular disease and yet they may indicate neither the one nor the other. Whenever there is mechanical impediment to the circulation from valvular disease, there is in almost every case a *murmur* audible. But there are murmurs without the valvular impediment, yet not without cause; but this cause is not always an impediment to the circulation.

23. Take, for example, those cases in which the murmur comes and goes, as it does sometimes in convalescence from rheumatic endocarditis. Here the murmur doubtless depends on some obstruction at the orifices of the heart which is so

nearly removed by the process of recovery as to cause a murmur only during exertion—during increased action of the heart; while directly the patient is in repose and the heart has had rest, the murmur vanishes, to recur upon subsequent exertion, and finally to vanish altogether when the patient is well.

24. A patient has uneasiness about the heart; it is even painful. The pulse is accelerated; the impulse of the heart is increased; the sound louder: nothing more; or, perhaps the patient says he gets fatigued sooner than formerly; no murmur is heard on auscultation, if the patient have been some moments in repose; but if he be directed to swing his arms violently, or walk briskly, and the ear be then applied to the precordia, endocardial murmurs are immediately manifest, and gradually vanish, even while one is listening. Now, here then is possibly the beginning of unsoundness of the heart, and possibly the murmur is the result of some alteration, of a temporary character, in the quality or quantity of the blood relatively to the dynamic condition of the heart at that particular time, or during a period of one or more days. Should the murmur continue, that is, become persistent, even though it continue of the "come and go" character, it is

an organic murmur, simply because inorganic murmurs from spanæmia, or merely dynamic changes in the heart itself, are in the very nature of the proposition not persistent, because their cause is not persistent, neither in its existence nor in its consequences.

25. There are cases in which a systolic murmur is developed immediately before death ; and in the absence of *post-mortems*, it is impossible to say whether such murmurs are the result of inflammatory action—pericarditis—set up at that moment, of coagulation of the blood near the pulmonic or aortic orifices, of coagulation in the mitral orifice, by which its valves are rendered incompetent, of spanæmia, or finally of dynamic alterations in the heart itself, occurring immediately before dissolution ; and the inquiry for the cause is unprofitable, for the patient dies, and the murmur is a mere declaration that death is imminent, and no longer to be warded off.

26. A murmur is easily developed in children, by pressure of the ear, or of the stethoscope, when there is no cause of a murmur *within* the body. *a.* A murmur may occasionally be developed in adults in the same manner, but in such case, the practised ear will easily distinguish the false from the true murmur by its quality. *b.*

Chicken-breast, though scarcely a deformity, may so displace and impinge the heart as to develope a murmur. Increase of area of precordial dullness to percussion, and extensive precordial impulse, are diagnostic signs of hypertrophy. Yet both these signs occur in chicken-breast ; and if to these be added a systolic murmur, we have the complete signs of hypertrophy with valvular disease ; and yet there is neither the one nor the other. In this case the murmur is easily produced, if absent, by the pressure of the stethoscope.

c. The respiratory murmur can be excluded as a source of error, by directing the patient to hold his breath an instant. *d.* Latham says a murmur is often heard between the upper edge of the second and the lower edge of the third left costal cartilage, and an inch along and between each of these ribs the murmur is systolic bellows-like, unmistakable in character. At the same time it is heard nowhere else in the heart, or arteries, or lungs. It occurs in phthisical patients, or in those who are justly suspected of being so. Whether this murmur proceed from the pulmonary artery in its first division, in consequence of its own disease, or in consequence of pressure upon it from consolidated or altered lung, is unknown.

Whenever there is suspicion of tuberculosis, the presence of this murmur will tend to confirm it; otherwise, it has no known value.

27. There is also the well known anemic murmur. These murmurs may be present not only in typical anemia—aglobulia—that is, defect of red blood globules, but also in hemorrhagic anemias, in typhus, in cholera, in all exhaustive affections, and in almost every variety of spanæmia.

28. Just how depravity of the blood causes murmurs in the heart, we cannot say; but the fact remains; and so soon as we remove the blood disease, the murmur disappears and returns no more. The anemic murmur cannot be confounded with that of unsound valves, on account of the accompanying symptoms. These are overwhelming in number and obviousness: "the surface pale and cold, palpitation and dyspnœa, appetite perverse, digestion imperfect, nutrition insufficient, secretions scanty and unhealthy, pain everywhere, a shattered nervous system and an enfeebled brain." These are not symptoms of unsound heart, but almost peculiar to anemia.

29. It has been considered a law, that a diastolic *murmur* proceeding from the heart is *always* an organic murmur, and that if it take the place

of the *second* sound, it is of valvular origin—semilunar—but there is reason to believe that a diastolic murmur may be generated in certain dynamic conditions of the heart, depending upon defective innervation, through *particular* nerve filaments, by which the function of a segment of a valve might be temporarily impaired so as to render the whole valve incompetent for a limited period. A clot might also so entangle a valve as to prevent its closure for a time, and yet eventually be all washed away and carried into the torrent of the circulation, either in complete solution or as emboli, to cause obstructions in remote organs. Walshe says that he has met with some few instances of inconstant murmur-like quality of the diastolic sound at the base, which appeared to him to be possibly dependent on disordered dynamism of the aortic valves. "It remains for future inquiries to determine, also, whether temporary reflux, with its murmur, may not be caused by dynamic imperfection, without structural change in the valves."

30. Those hæmic murmurs that characterize aneurisms, or indicate pressure on arterial or venous trunks, by abnormal growth, or foreign bodies, either in or out of the body, are excluded from consideration in this estimate of inorganic murmurs.

There is little doubt that one accustomed to auscultation of the heart would distinguish an inorganic from an organic murmur, in almost every case, by the mere quality, pitch, tone, strength and duration of the murmur alone.

CHAPTER VII.

PERICARDITIS AND ENDOCARDITIS. ENDO-PERICARDITIS—CARDITIS.

31. By inflammation of the heart's membranes, is understood something altogether different from "disease of the heart." The latter phrase is vulgarized in and out of the profession so as to mean the concrete condition of the heart in a state of chronic, not acute, unsoundness. Inflammation of the heart's membranes is a phrase the import of which would be understood by the profession, and the use of which is limited to the profession. It is divided into two varieties, one pertaining to the inside—endocarditis—the other to the outside of the heart—pericarditis. These two affections originate in similar though not continuous membranes; the one, endocarditis, attacks the lining membrane of the heart's cavities; the other, pericarditis, attacks their investing membrane—the sack in which the heart is enveloped.

a. Omitting details of great interest, but not absolutely necessary to accurate diagnosis, these diseases are distinguished by signs so exclusively characteristic, that if the heart's membranes are inflamed at all, it can generally be made out whether the affection is one or both of the varieties mentioned.

b. The characteristic sign of endocarditis is an endocardial *murmur*, systolic in time, occurring suddenly in a person with fever or acute rheumatism, in whom it had never occurred before. One can never be absolutely certain of the endocardial inflammation, until the *bellows murmur* is heard, no matter how great the pain in the heart, how great the palpitation, how irregular, or intermittent or unequal the pulse, or how hot and livid the skin. Treatment of course should not be postponed until the murmur be heard, neither can it be positively said there is no endocarditis before the murmur is heard; but, on the contrary, it may be *reasonably suspected* without the murmur, but not positively made out. *c. The murmur is essential to the positive diagnosis.* (Latham.)

d. The murmur occurring for the first time, during the presence of constitutional inflammatory symptoms, determines that the general inflammation has touched the endocardium.

e. The seat of the greatest intensity of the murmur determines—in limits—the chief seat of the inflammatory process. The murmur is usually heard loudest at those parts of the thoracic walls which are nearest that portion of the heart from which the murmur originates. A systolic murmur may be heard over the right or left ventricle, or over the aorta alone, or over the pulmonary artery, or over two or more of these parts at the same time.

f. The sounds may be normal, or louder, or duller, or more indistinct, or shorter than natural, and at times they may be almost inaudible, from the weakness or temporary restraint of the heart's contractions ; or, in consequence of great effusion in the sac of the pericardium, when that membrane is also affected.

g. The valves are sometimes rendered incompetent for a few hours or days only, and during this time a murmur of regurgitation—mitral—may be heard at left apex, or a diastolic murmur may be heard over the third left cartilage, and thence down the sternum from incompetency of the aortic valves.

32. If the *pulmonic* valves should become incompetent, (a thing which scarcely ever occurs,) a diastolic murmur would be heard, loudest in *second* (pulmonic) interspace, and thence directly

upwards. The *aortic* diastolic murmur is not infrequent after the first few days of endocarditis.

33. *Pericarditis* is not known to be an idiopathic disease. It is chiefly the result of acute rheumatism; rarely of Bright's disease of the kidneys. It may attend or directly follow any of the exhaustive diseases—as typhoid fever, variola, scarlatina, diphtheria, pyæmia frequently, (one in seven cases of pyæmia have pericarditis, but pyæmia itself is very rare); it is sometimes associated with pleuritis, or pleuro-pneumonia, but has no known dependency on these lesions; with albuminuria from any cause—wherever there is albuminuria, the heart should be watched for endo-pericarditis. I have seen it with scurvy in three instances in the late civil war; with influenza, erysipelas and consumption; hepatic abscess of left lobe—a case observed by the author at Fort Yuma in 1861, verified by *post-mortem* examination—very rarely with cancerous and tuberculous diatheses. Of course it may be produced by any direct injury to the pericardium, as wounds, blows, fractured ribs, etc.

It is more likely to follow or attend rheumatism in youths than in adults; in females than in males; hence it is more frequent in these classes of persons than in adult males. Soldiers are more liable to scurvy than civilians, hence

they are more liable to pericarditis and endo-pericarditis from this cause.

a. Its most *obvious* symptom is the anxious and drawn expression of the features, and the absolute immobility of the patient, as to voluntary muscular motions; but this symptom is not always present, and when present is also a sign of pleuritis.

b. The characteristic, the *diagnostic* audible sign is a *friction* murmur, or rather a friction sound. If one attempts to describe the abnormal murmur he hears in pericarditis, it will be by some similie into which enters the friction of two surfaces of a greater or less degree of moisture, or dryness, or roughness. It would never occur to imitate the *friction* sound of *pericarditis* by modifications of the respiratory act, while it would never occur to imitate the "bellows murmur"—"*bruit de soufflet*" murmur of endocarditis in any other manner except one called in the aid of external atmospheric murmurs.

The *friction* sound may be loud or low-toned, sharp or dull, single or double, but it always suggests the idea of attrition, and never that of expiration or inspiration, or blowing; and so the contrary.

b. The chief local symptom of *pericarditis* is

pain (Walshe); but the same author says that in the majority of cases pain is either absent or of slight severity.

c. The pericardium is peculiarly insensible when inflamed, in Bright's disease, and patients often persist in denying the existence of symptoms referable to the heart; but in these cases pressure with the flat hand over the heart will cause a degree of uneasiness which is intolerable, although it may not be precisely pain; and this insensibility is not confined to pericarditis in Bright's disease, but occurs especially in asthenic pericarditis. Hence the "chief local symptom" (pain) avails little in diagnosis, much less than immobility and lying on the back, the anxious look, attendant rheumatism and inflammatory febrile pulse.

d. The distinction between endocardial *murmur* and exocardial *friction* is occasionally impossible. (Walshe.)

e. There may or may not be bulging of the precordia from pericardial effusion. There may or may not be friction—*fremitus*—appreciable to palpation. The heart's impulse is generally greater in extent but not in force, and may even be much weaker than natural.

h. Pericardial effusion may develope apparently endocardial systolic murmurs, by pressure on the

roots of the great vessels, thus leading to the suspicion of endocarditis, when the endocardium, valves and vessels may be entirely healthy.

34. It is believed, also, that pleuritic effusions, in very rare instances, so contort the vessels at their orifices as to produce incompetency of valves, thus causing diastolic murmurs heard even at the apex and down the sternum, the peculiar seats of *organic regurgitant murmurs.*

35. I recently saw, for a few moments, during my first and only visit, a case of *pericarditis*, remarkable for the rapidity of its progress and for its fatal termination. When I saw the patient, he was suffering with frightful dyspnœa, which was supposed by his attendants to be "asthma, aggravated by wind on the stomach"—[There was epigastric fullness, which is not uncommon in pericarditis, second stage]—for which they were administering stimulants and nauseants, in bulky doses, to cause emesis; the patient was held, sitting upright, although he desired to lie down; his skin was livid and bathed in viscid perspiration; his pulse was exceedingly rapid, small, irregular, intermittent, unequal; his extremities cold; his voice reduced to a whisper; deglutination, even of fluids, difficult. Percussion of the precordia was impracticable, but during the instant I listened at the

heart, I heard a dull, wet* second sound, (no first sound,) and a faint murmur, systolic in time—no *friction* sound. My diagnosis, to my own mind, was rapid but not exhaustive: pericarditis, with either very considerable effusion, incompetent mitral valves, or fatty heart with incompetent mitral valves. The fatty heart was rendered doubtful, by the persistence of the pulse, which in the last hours of this affliction is generally absent, or answers to the systoles perhaps ten or twenty times only in a hundred. The absent first sound was as accountable on the supposition of effusion, as of softened heart;† hence, on the pulse alone, I excluded the latter. I had the melancholy good fortune to assist at the *post mortem*, some forty-eight hours later. The left ventricle was generally dilated; the mitral valves were incompetent from analogous deposits; the beginning of the aorta was atheromatous; there were some patches of lymph on the visceral duplicature of pericardium, and very considerable serous effusion; amount not measured, but supposed to reach eight ounces. The proper tissue of the heart was softer than natural—not examined microscopically.

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* A sound like that of striking a table with a wet napkin.

† See Appendix F.

The duration of this case might have been indefinitely prolonged, had the serious damage to the heart been recognized at the beginning instead of the end of the last week of life. A recumbent position, aconite and veratrum viride in minute and alternate doses, saline evacuants,* saline diuretics, leeches and cups at the precordia, sinapisms to the knees, ankles and elbows, suspension of food, except beef juice in small quantity, no stimulants, properly so called, except on the special indication of failing pulse and threatened syncope; then, brandy, or ammonia and lavender. With this treatment from the beginning, it is probable that the pericarditis would have terminated in complete recovery; the mitral disease being slight, would have been harmless for the time; the fatty degeneration, if certainly present, which was not proved, would not necessarily have caused death so soon.

36. There seems to be great discrepancy in authors, Latham and Skoda, for example, concerning the distinctive characters of *friction sound* (exocardial) and *bellows murmur* (endocardial).

37. To my mind it is clear that a sound whose similie is *friction* must in the natu re of the proposition be dissimilar from one whose similie is

* See Appendix G.

murmur; and this is the precise actual difference between the two murmurs. They are recognized by distinct names, *friction murmur* and *bellows murmur*, all through the literature of diseases of the heart, from Corvisart to Walshe.

38. WALSHE and others say, the murmur, when there is doubt of its extra- or intra-cardiac origin, may be identified as *exocardial*, by being more superficial and more diffused than *endocardial murmurs*. Skoda says flatly that in his opinion they cannot be so distinguished. Boillaud experienced great difficulty in discriminating attrition and valvular murmurs.

39. HOPE makes the distinction by no means difficult. The existence of the two classes of murmurs is indubitable, as all writers on the heart, since Collin, in 1824, distinguish them as being referable; the *valvular murmurs* to the *endocardium;* the *attrition murmurs* to the *exocardium.*

40. SKODA recognizes the existence of the two classes of murmurs. After acknowledging the *new-leather* attrition sound, *which is very rare*, the rustling and rubbing sound (frequent), the *scratching* sound, from bony, chalky, or fibro-cartilaginous concretions, rubbing against each other, or some part of the pericardium during the heart's movements, he says: "There is no doubt that all

these different friction sounds of the pericardium have a real existence. * * * * * According to my own experience, there is no kind of endocardial murmur, with the exception of the whistling, which may not be imitated by a friction sound of the pericardium ; and no pericardial murmur which may not resemble an endocardial murmur."

41. HOPE and MARKHAM believe the distinction of the *murmur* from the friction not difficult ; the former did not find it difficult to discriminate between *attrition sounds* and *murmurs*, "even when the two classes of sounds existed simultaneously and each was double." The former discriminates by listening for the *murmurs* of the semi-lunar valves two or more inches up the aorta or pulmonary artery, where *attrition* murmurs are generally inaudible ; and by listening to the murmurs of the auriculo-ventricular valves, a little above the apex of the heart, where they are sure to be loudest, whereas, *attrition* murmurs may be louder at other parts of the heart where they happen to be generated.

42. He further distinguishes them by the following peculiarities :

a. Attrition murmurs have usually a much

rougher quality of sound than the valvular murmur, so that when the two co-exist, the one may be heard *through* the other.

b. When a murmur with the second sound is rough, as rasping, creaking, croaking, etc., it is certainly from attrition.

43. *Second sound valvular murmurs*—diastolic murmurs—are never rough, as the diastolic currents are in the nature of the forces causing them, too feeble to produce rough murmurs. A diastolic mitral murmur is faintly audible; a diastolic aortic murmur is louder, but still smooth. Diastolic pulmonic murmurs almost never occur. Systolic tricuspid murmur is known by its smooth blowing quality, and by the attending jugular pulse.

44. "Attrition murmurs are almost always attended by vibratory tremor." This "tremor" I have never been able to verify, even where pericarditis was undoubted before and verified after death.

45. MARKHAM says: "a *fremitus* is said to be sometimes felt over the precordial region when the hand is laid thereon." Skoda places no reliance upon this sign. On the contrary, Latham relied greatly upon it as a distinctive sign. He has often seen an undulatory motion between the cartilages of the second and third ribs, or be-

tween cartilages of third and fourth ribs, left side, during the dull percussion and exocardial murmur period of pericarditis. He also often *felt* vibratory motion in the same interspaces, *second* and *third*, and nowhere else. The undulatory motion and vibratory motion often occurred simultaneously, but he thought the vibration the more frequent. They were sometimes present between the cartilages of second and third, and third and fourth left ribs, simultaneously—*tactile fremitus.*

46. WALSHE says it is rarer and more migratory and superficial to the touch than valvular thrill, distinguished from pleural fremitus by causing the patient to hold his breath ; not implicitly reliable, because its mechanism is unknown, and it *may* arise from causes external to the pericardium, and be present during suspension of the breath. But, if present with the exocardial *friction*, it is a valuable aid in the diagnosis.

47. STOKES says, attrition murmurs are apt to undergo frequent and sudden changes of character and of situation which are very pathognomonic, because valvular murmurs change little in character, *and not at all in situation.*

48. This last, though agreeing with Walshe,

Hope and others, is very discrepant with Latham, who never observed the fremitus, except in the *second* and *third* left cartilage interspaces. Can it be possible so careful an observer as Latham mistook a *pulmonic valvular thrill* for a pericardial *fremitus ?*

49. Finally, concerning the murmurs. It is probable they are distinguishable by most observers—the *attrition* from the *valvular*—or that the cases in which they are undistinguishable are exceedingly rare, or that there are cases in which both classes of sounds occur, each being modified by the other, so that each becomes unrecognizable for a longer or shorter period ; but patient and repeated observation would in the end discriminate in almost every case between an attrition and valvular murmur.

50. But it is of no vital importance whether the distinctive diagnosis be made at all, for the two diseases are essentially similar in immediate effects, and in indications of treatment ; and the indications of treatment are by far of greater importance than any theoretical considerations of the distinctive characters of the two diseases.

51. *Pericarditis* is a much less frequent disease in warm than in cold climates, other things equal.

It is by no means so frequent in San Francisco as in Edinburgh or London.

52. In more than a hundred cases of rheumatism treated by me in Arizona and Southern California during the years 1862 and 1863, I saw but one in which I was able to verify that the inflammation had affected the heart; yet in every case I sought diligently and frequently the symptoms of rheumatic carditis.

The following is from my notes of this case taken in the field.

Case of Acute Endocarditis, apparently idiopathic, with Emboli.—Argo, a soldier, aged 25, tall, fair-haired, muscular, came on the sick list for a cough. Ordered brown mixture—he did not seem much sick.

Second day. Argo breathed short; had fever in the evening. Ordered saline purge and quinine, five grains with half a grain of opium, when his fever should go off; continue cough mixture. He had no crepitation in chest; some few rather coarse rales; no pain in the chest, but coughed severely at times; little or no expectoration: respiration 45; pulse 110, feeble, small.

Third day. A. has an anxious look face cadaveric and perspiring; appearance of great agony,

but he is able to walk; his respiratien 45; pulse 90, very weak and small; nails, ears, lips, etc., livid; says he has *no pain*, but feels "tight here," placing his hand on the sternum, at its junction with fourth and fifth ribs.

Auscultation revealed no abnormal sounds in either of the lungs; over some portions of the lung respiration scarcely audible, and in other small portions, no air entered.

The heart's impulse, at the fifth space to the right of left nipple, very feeble; sounds are exceedingly weak. There was something indeterminable and undefinable about the quality of the stytole, and this with the tightness over the base of the heart, made me uneasy about the result. Ordered cups and sinapisms over the precordia, syrup of senega for the cough, and beef tea for diet. At four A. M. he died, without any of the nurses in his ward observing him until he was quite dead. They heard a very "wheezy breathing" a few moments before he was found dead.

Post-mortem at nine A. M. same day. On opening thorax, anterior aspect of lungs appeared natural; no adhesions of either lung; no tubercles. On lifting out lungs, lower lobes of both were found congested posteriorly, and very dark,

with fluid blood; upper lobe of right lung congested posteriorly.

Size of heart and external aspect normal; no effusion in pericardium; no lymph patches, no white spots, nor congested coronary vessels. On removing and washing heart, in both right and left ventricles were found pale clots, like strips of washed muscle wound and interlaced with fleshy columns and tendinous cords, and under the valves. The right auricle was completely plugged with a semi-organized looking clot, which I peeled out from its matrix, as a false membrane peels out from the sinuosities of the fauces. These clots, after being removed and thoroughly washed of all their soluble portions, weighed ninety grains. The endocardium presented numerous signs of inflammation, especially about the auricles and valves; left auricle contained no clot; valves were competent, and no orifice was appreciably constricted. These clots must have been formed before death, which evidently occurred the moment the right auricle became so filled with the clot that no more blood could enter and pass through it from the *venæ cavæ*.

The man lived four days after he first complained of "tightness in the chest." At the time

of this occurrence, there were many cases of rheumatism in camp, which was attributed to sleeping on the green salt grass, with only one blanket. I do not think this was a case of simple embolism from the accidental development of the coagulating principle * of the blood, and I am sure it did not result from fatty degeneration of the heart, by which it was rendered incapable of emptying its cavities, for the heart's substance was firm, and normally free from fat.

54. During the last four years, I have seen several cases of pain and uneasiness about the heart during the course of acute rheumatism. In one there was great anxiety of expression, and extremely small and feeble pulse, which made me fear carditis and the formation of *emboli*. I gave in this case whiskey and water, alternated with fluid extract of digitalis, in minute doses, every hour, without interrupting the usual rheumatic treatment with propylamin and laudanum. The patient recovered.

55. The treatment of pericarditis and endocarditis, or the two conjoined, is essentially the same.

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* On Embolism, see Appendix H.

56. The diet must be diminished in quantity and in blood-making quality, if the disease, as most frequently happens is of acute rheumatic origin—in other words, if it is *sthenic* in quality.

57. Blood *may* be taken (if the patient be full-blooded, or in good blood-making condition,) from the arm, to the extent of a pound, early in the attack, the earlier the better, and this may be followed by five, ten or twenty leeches (according to the violence of the attack and condition of particular case,) to the precordia, and these may be followed by compresses, wrung out of hot water, laid over the leech bites, not merely to promote the flow of blood, but to alleviate the precordial anxiety and tightness of the chest, which will be thus accomplished in a surprising manner.

58. The dose of propylamin, providing we are treating the rheumatism with propylamin, may be quadrupled immediately.

59. If the heart beats more than 90, and especially if its beats are not rhythmical, half a drop of fluid extract of digitalis and five drops of McMunn's elixir of opium may be given every hour, until pulse falls to 75, and increases in size and regularity. Syncope must be guarded against instantly and hourly.

60. It will rarely be found *necessary* to bleed at the arm at all.

61. Leeches, scarifications and cups to precordia, sinapisms and friction to the joints, a blister around each knee or elbow joint, with a brisk and certain purge, (calomel fifteen or twenty grains at one dose, is the best,) followed by saline diuretics, combined with minute doses of tartrate of antimony and posassa, (say the thirty-second part of a grain hourly,) hot bottles to the loins and feet. Some or all of these means, or others on the same principles, will, in most cases, be found quite sufficient to mitigate the attack, and conduct the disease to its usual result, namely, termination in recovery.

62. It is very rare that rheumatic pericarditis or endocarditis treated rationally, or let alone, terminates fatally; but it is altogether probable that the worst consequences—chronic unsoundness of the valves from endocarditis, or pericardial adhesions from pericarditis, maybe prevented altogether, or greatly modified by rational treatment.

63. In the nature of the case, this theoretical probability can never be demonstrated; for, if some time after, a case of marked endo-pericarditis, treated rationally, and apparently entirely cured, should die of some other affection, and the

heart should be found intact, it would not follow that the treatment had saved it; and were it found chronically diseased, it would not follow that the treatment had been defective, excessive or erroneous.

64. But, if a patient of mine should die apparently of rheumatic pericarditis, I should doubt my diagnosis, or be dissatisfied with the energy or rationality of my treatment. Not so with the asthenic forms of pericarditis; these are much more likely in their own nature to be fatal from the beginning.

65. In *asthenic* rheumatism, no matter from what source the asthenia springs, pericarditis should not be treated antiphlogistically. Carbonate of potash and of ammonia internally, counter-irritation, good nourishment and moderate doses of wine or brandy, form the staple agents of treatment. (Walshe.)

66. I would caution, in the use of alcoholic stimulation, to watch its effects constantly. The pulse should not be carried above 85; on the other hand, it should not be allowed to fall below 70. It can be maintained in these limits generally, (except when the pericarditis accompanies renal degeneration,) with small doses of digitalis and laudanum.

67. Alkalies—bicarbonate of potash, liquor potassæ, are theoretically if not practically worthy of trial in very large doses in the fibrinous diathesis, especially if there be any suspicion of emboli in the heart or arteries. I need not say these remedies are to be largely diluted.

68. If a stimulant is required, carbonate of ammonia, or aromatic spirits of ammonia and tincture of lavender * may be given in conjunction with the alkalies (69).

69. The enormous doses of brandy, a pint or more a day, recommended by Todd and Bowman, are repugnant to theory and prohibited by unprejudiced clinical experience. *a.* Can any one doubt that intoxication induces venous congestion of the corronary veins, as of others?

Can any one doubt that congestion of the heart and lungs increases the labor of the heart (already requiring alleviation,) and thus diminishes its dynamism? Is it supposed that impaired dynamism of the heart is conducive to the restoration of its inflamed serous envelope?

70. It cannot be denied that brandy diminishes the coagulability of the blood, and thus would tend to prevent the formation of intra-ventricular clot

* Sp. ammon. aromat. dr. i; sp. lavend. comp. dr. vij. M. Sig. Dose a teaspoonful as often as required, in water.

in endocarditis; but so does chloroform diminish or prevent coagulability; so does electricity and many poisons, but we cannot deduce from this one fact, their therapeutic utility.

71. We know that alcohol given in large doses to sthenic or plethoric patients increases their fever, adds to their irritability, congests the liver, impairs pulmonic functions, diminishes the action of the skin, and does not always act on the kidneys; that it impairs appetite and assimilation; that it is unquestionably objectionable in Bright's disease, even in not very large doses.

72. I do not object to brandy in any disease, but I cannot help thinking that it ought to be used in much smaller quantities in acute affections than recommended by the distinguished authors just mentioned.

73. Patients with any form of organic affection of the heart bear brandy very indifferently, and generally object to it very strongly, from a firm conviction that it makes them worse. Besides, in organic affections of the heart, acute or chronic, the liver is rarely exempt, and all drinkers know the antipathy of this organ, whenever so little disturbed, to large doses of brandy or whiskey. Soda-water suits it much better.

74. It seems to me, the general experience of mankind should be somewhat regarded in reference to the commonest articles of daily use, and this general experience is opposed to excessive employment of the hydro-carbons in acute and inflammatory diseases.

75. *Recapitulation.*—*a.* The treatment of pericarditis and endocarditis is nearly the same. *b.* The latter is more injurious in its consequences, and hence requires more care in treatment. *c.* There are two forms: the *sthenic* and *asthenic* of both lesions. *d.* The *sthenic* is treated *antiphlogistically*, in the rational sense, by which neither salivation nor exhaustive depletion is understood; that is, it is treated with aperients, watery diuretics, alkalies, digitalis, opium, propylamin, counter-irritation, heat locally applied, local depletion, general depletion to relieve a pressing need, but not to arrest inflammation, etc. *e.* The *asthenic* is treated on general principles, specially applied. *f.* Hæmatogens — beef-juice, egg-nogg, vegetable alkaloids, bromide of potassium, perhaps cod-liver oil—counter-irritation and frictions, dry cups to the precordia, arterial and nerve stimulants, nutritious diet.

Mr. Ure (Braithwaite's Retrospect, vol. 1, p. 846,) proposes the use of *sulphate of manganese*,

not omitting but supplementing the alkaline treatment of rheumatism, to prevent it attacking the heart.

M. Gemlin ascertained that this remedy augments remarkably the secretion of bile, and it has often been noticed that when uric acid increases, bile decreases, and *visa versa*. Hence, if we increase the bile, we diminish the rheumatism. Mr. Ure recommends a drachm of this sulphate to be dissolved in half a pint of water, and swallowed before breakfast. It will be followed by one or more motions, of biliary description.

f. This remedy is worthy of trial on theoretical grounds, especially as it is harmless.

g. Nitrate of potash has been much recommended, on account of its property of dissolving fibrin.

h. It may be given because it is an alkali, to prevent the tendency of the blood to become acid, and also as a solvent of fibrin, to prevent the precipitation of this material on the valves of the heart and other portions of the endocardium. It is probable that the fibrinous matter found on the valves of the heart is produced *both* from inflammation of the lining membrane and from precipitation, when it is in excess in the blood, and when the circulation is very much impeded

or very much slowed, even for a moment, from any accidental cause. I am aware that stasis alone will not induce coagulation, but it is not denied that coagulation is thus hastened, under favorable conditions. *

i. Opium is often needed in enormous doses, to produce any sensible effect.

j. As a diuretic in pericarditis in Bright's disease, I should expect great benefit from gallic acid, in from thirty to sixty grains at a dose in as much water as the patient could conveniently drink, four or five times a day. The hot-air or vapor-bath is especially recommended as a sudorific in this variety of pericarditis, because it can be administered in bed, without disturbing the patient and without raising the head, which often causes great prostration.

k. Purgatives are to be employed with great caution, in either the *sthenic* or *asthenic* forms, but especially in the latter. Suphate of magnesia should not be employed.

76. The *chronic* forms of these lesions are to be treated on general principles, it being understood that they can be alleviated, but not cured.

77. For example, if life is in imminent jeopardy

* See Appendix H.

by the accumulation of fluid in the pericardium, there can be no question of the propriety of tapping near the base of the heart, upper angle of fourth left interspace, and re-injecting a small quantity, of equal parts, tincture of iodine and distilled water, at the temperature of the blood, before removing the canula of the trocar. Iodine injections are recommended by Aran. This iodine liquid has been proved harmless to serous membranes in numberless examples.

a. Seton at the præcordia, mercurial inunction, iodine ointment, or dilute tincture of iodine are seemingly beneficial.

78. Blisters applied near the præcordia might promote absorption.

79. A dry nutritious diet and diuretics are recommended, with great reason. Iodide of potassium taken internally could not retard absorption of lymph and serum, and by many is supposed to materially aid this process. But I have never observed such a result either in this or allied effusions.

80. Here passive exercise in the open air, and even sea voyages may be advised, with prospect of alleviation and arrest of the slow inflammatory process. The trial of alkaline springs should be made, if possible.

a. It should be borne in mind that the *prognosis* as to fatality is more serious in *pericarditis* than in *endocarditis.* The latter is rarely fatal, whereas the former terminates in death with sufficient frequency to have induced M. Louis to estimate the deaths from this cause at one in six of the cases attacked. Walshe had seen only seven or eight fatal cases of fatal rheumatic pericarditis. Death rarely occurs in pericarditis "before the tenth or twelfth day." It may occur, however, within thirty-six hours, or be postponed some weeks.

b. We have also chronic forms of both these affections, but from what has been already said of the acute forms and of valvular diseases, the *treatment* of chronic endocarditis is obvious, and that of pericarditis requires but mere mention. The hypertrophy and dilatation of the left ventricle attending chronic pericarditis are less serious than when these affections are the result of valvular disease; the treatment of these conditions of the heart will be considered in a subsequent chapter. As for the rest, the treatment is to be directed to the absorption of the effused fluid, and to fortifying and controling the action of the heart. It is doubtful whether local applications to the præcordia will be of much benefit, except for the modification of reflex symptoms;

but most of the discutient class of unguents, tinctures and liniments, as well as blisters, setons, cauteries, etc. are still recommended as of possible utility. In chronic pericarditic effusion, paracentesis of the pericardium, as already suggested, is unobjectionable, after a reasonable trial of other remedies.

c. Acute and chronic endo-pericarditis can be appreciated and treated by the principles already suggested.

d. Acute carditis is not susceptible of diagnosis in the present state of knowledge; that it has a real existence is well proven by *post mortem* observation, but it has no known clinical signs by which it can be discriminated from *endo-pericarditis.* Hence, it is obvious its separate treatment it not a subject of rational consideration. Fortunately it is a rare affection. Similar remarks are applicable to the *chronic* form of *carditis.*

e. Hemorrhage within the proper substance of the heart does occur, but we have no diagnostic symptoms. So effusion of the blood into the pericardium— *hæmo-pericardium*—and under the endocardium — *hæmo-endocardium* — are without diagnostic symptoms of any value. Œdema of

the heart exists, but cannot be diagnosticated in the present state of knowledge.

f. HYDRO-PERICARDIUM is recognizable, if the dropsy be considerable by the pyramidal outline of præcordial dullness, the same as in the effusion stage of *pericarditis.* (The apex of the dullness being above its natural point, and rising to the second or perhaps to the first cartilage, while the *base* does not fall proportionally, indeed not at all, but remains at the lower margin of the sixth rib.) The visible impulse is undulatory, unless the effusion be slight. A sensation of oppression at the præcordia is often though not always felt; the pulse is not affected. The dropsy must depend on some altered condition of the blood, conjoined with some other organic lesion; hence the general symptoms will be those of the subjacent affection, and the *treatment* will be the same as that of other dropsies, though diuretics and cathartics will be found less efficient. Dry cupping, blisters and electricity, one or the other, may be applied at or near the præcordia with considerable hope of benefit.

g. Paracentisis, and subsequent iodinized injections, so as to excite adhesion of the pericardial surfaces, may be found advisable under particular circumstances; but the principal hope of cure

lies in the alleviation or removal of the disease on which this effusion depends.

h. The *diagnosis* and *treatment* of cases in which air or gasses find ingress to the heart's cavities or beneath its membranes cannot be rationally considered, and hence the treatment must be suggested by the special indications, in any suspected case.

i. Cardiac *atrophy*, whether limited to the muscular walls or valves of the heart, is clinically unrecognizable, and an anatomical curiosity, and may be clinically ignored.

j. Hypertrophy of the valves of the heart, would, at first view, seem to be the result of endocarditis, but it may rationally result from excessive function, and consequently excessive nutrition, as a result of hypertrophy pure and simple. It cannot be recognized clinically, and if suspected its treatment is merged in that of *hypertrophy*.

CHAPTER VIII.

HYPERTROPHY. DILATATION.

81. *Hypertrophy* is not very frequently an idiopathic disease. Like dilatation, it is generally the outgrowth of some previously existing lesion of the valves or vessels proceeding from the heart; of some impediment, (as of a tumor or a consolidated lung,) to the free movement of the heart.

82. It may be caused by excessive nourishment, too free living, and excessive drinking, excessive labor in which the strength is taxed to the utmost. Gymnasts, stevedores, furniture-movers, iron-moulders, athletes, all furnish examples of pure hypertrophy. It is comparatively rare in females, except in those who have borne many children.

83. Obstructions in the lungs would cause hypertrophy of right sooner than the left ventricle.

84. The hypertrophy attending free living and sedentary habits is apt to be attended with fatty metamorphosis, and is of much more dangerous significance than hypertrophy pure and simple from excessive function.

85. The left ventricle is by far the most frequently affected ; next, the left auricle, then the

right ventricle; the right auricle is very rarely hypertrophous.

86. *Left ventricle* in a hypertrophous state is recognized by præcordial bulging, widened interspaces, heaving, sustained impulse, when the side of the observer's head is laid over the heart; regular rhythm, force of impulse unequal; extent of, increased; maximum of, about the left nipple.

a. Unless there is associated dilatation, the apex beats will rarely extend to the left of left nipple, or below the seventh rib. *b.* If there is dilatation also, the apex may be carried much farther beyond the nipple, and as low even as the eighth rib. *c.* In this case the visible impulse will be much greater, more knocking, heard distinctly and clearly some inches from the thorax; the impulse is also less heaving; the heart merely strikes, without raising the ribs and interspaces, and the whole præcordial surface, down as far as to the left of and below the left nipple, seems agitated in extreme case of dilatation with hypertrophy.

87. In the case I have in mind, Mrs. D——, of San Francisco, a young married lady, the thumping of the heart could be heard several feet, and the appearance of the impulse was that of the skin over the emaciated thorax, being raised in

numerous places in a sort of undulating rhythm, suddenly and a hundred and twenty times a minute. This was a case of enormous eccentric hypertrophy, and terminated fatally a few days after my first and only visit.

88. Dullness to percussion may extend from the second to the eighth rib, and from two inches to the right of the sternum to three inches outside of a vertical line drawn through the left nipple. In exceptional cases, "the percussion sound may be toneless and high-pitched all over the left lateral regions of the thorax, and vocal fremitus null, just as if pleural effusion existed." (Walshe.)

89. The *diagnosis* of hypertrophy, without or with marked dilatation, is not difficult. *a.* Besides the percussion and auscultory signs mentioned, there is in hypertrophy the marked symptom of persistent force of the pulse.

b. The pulse may vary in the course of a single day from 65 to 110, and from being full, hard and resistant, to being compressible, full and soft—rarely irregular or intermittent ; and when these conditions are present, other lesions exist in the heart. *c.* The patient will be strong, vigorous and full-blooded, of florid complexion, and apparently robust health. *d.* A case of this

kind now on my mind is a young man, aged 25, who can lift six hundred pounds, and who is in the habit of lifting two and three hundred at a time daily.

e. The strength is not lessened, but the dyspnœa becomes intolerable, from prolonged or excessive exertion. and cough supervenes.

f. The patient wants his head tilted forward when he sleeps, rather than have his shoulder raised with his head. There is no orthopnœa, but the head aches if lying in a line with the trunk. This symptom is not always present. *g.* The varying frequency of the pulse is doubtless exceptional in the case now under consideration. *h.* There is no dyspepsia, but little pain in the heart, but more or less uneasiness, and a sense of fullness ; frequently dizzy on stooping ; the conjunctiva habitually red ; sleep disturbed with strange dreams. [In this case the symptom was removed by thirty grains of bromide of potassium daily, at bed.time.] *i.* No increase of sexual desire in this case, as has been supposed on theoretical grounds. *j.* There is a systolic blowing murmur at base of varying loudness and not persistent. When the heart beats with unusual violence the murmur is very distinct ; when "slowed" and subdued with digitalis, it is

inaudible. *k.* I suspect this murmur is not rare, for water forced through a tube of varying calibre with considerable violence, emits a murmur at the point of variation in the diameter of the tube. *l.* The extremities do not swell, nor is there ascites. *m.* In this case there is frequent pain in the right side, and occasional congestion of the liver. *n.* The bowels are disposed to constipation ; nutrition and hematosis is excellent. *o.* There is no lividity of the nails, or ears, or lips; no coldness of hands or feet, except occasionally some hours after excessive fatigue. *p.* Dilatation may have begun in such a case, and be yet so slight as to be overshadowed by the hypertrophy.

90. The *treatment* of hypertrophy is directed to the retardation of the disease, probably to its arrest, possibly to its cure.

91. Few physicians now believe in the cure of hypertrophy. "On the other hand, it is not difficult to remove or greatly mitigate the symptoms of simple hypertrophy in the majority of cases, and render life, not merely tolerable, but comfortable."

92. The heart's action is to be quieted, and the amount of blood diminished without deteriorating its quality.

a. I have elsewhere (p. 61 *passim*) shown how this may be done, by diminishing the weight of the body, by means of restricted and dry diet. *b.* The heart must have less blood to impel, but it must have good blood for its own sustenance, or we may have fatty metamorphosis added to our hypertrophy. *c.* The patient may be cupped or leeched at the præcordia once a week, if necessary; purged occasionally; take diuretics most of the time—solution of perchloride of iron and digitalis, with antimony—baths every other day; plenty of passive exercise, until he is made quite comfortable, and all severe symptoms are removed. *d.* If the hypertrophy were caused by excessive exertion or labor, that must be stopped, of course; if by excessive eating and idleness, a restricted diet and some exercise must be enjoined, but much more caution in depletion will be required in treating this class of cases, than that in which the hypertrophy results from excessive function of the heart itself.

Case.—Mrs. J. S. consults me concerning bronchitis. She has taken all the nostrums and specifics, has been relieved often; sometimes has thought herself cured; but the annoying cough and frothy expectoration returns again and again, when she thinks she is nearly cured. On

listening at the præcordia, I hear a systolic murmur; move my ear to left apex and it is much louder; heart's impulse increased in extent and vigor. Apex beat farther to left than normal; the head is lifted by the throb of a hypertrophied heart; the sounds are nearer to the ear and more neatly accented than normally. She has incompetent mitral valves, dilatation with hypertrophy, the latter still in excess; turgescence of lungs from refluent current; mechanical compression of left lung from enlarged heart; hence initative cough, with increased mucous expectoration.

Give a calomel purge, followed by syrup of wild cherry and tincture of aconite; restrict diet one half. In three days the cough has disappeared, and the patient feels much better, though her garments fitting less snugly, she "*fears she is losing flesh!*"

She *is* losing flesh, and must never again carry so much flesh, if she hopes to be free from her "bronchitis." This is one case of a whole class in which loss is gain. A heart which will utterly fail to impel 16 or 17 pounds of blood—the normal amount—will get along with 14 or 15 pounds with little or no difficulty. It must not be inferred from this, that *therefore* bleeding to the

amount of one or two pounds will answer the purpose and prove beneficial. The weight of the body must be diminished in the same proportion. If we wish to abstract a pound of blood from a patient with dilated heart, we must do so by reducing the patient eight pounds in flesh, by means of low diet, or, in cases of urgent need, by total abstinence from food until the effect is produced. It is remarkable how soon the overtasked heart will recover its tone when the patient entirely abstains from food, and takes small doses of some nerve sedative to quiet the irritability which hunger sometimes induces. The pressure disappears from the chest, as weight diminishes, and very soon, say from three to five days, the patient, if not yet in the dropsical stage, will say he is better than ever, and that he scarcely knows he has a heart. Even now, a single full meal will bring back all his consciousness of his heart and of its unnatural condition.

93. Pure and simple hypertrophy is not apt to destroy life. *a.* It is only when other organic diseases have come on, or when dilatation becomes excessive that life is in jeopardy.

94. *Dilatation* will not be difficult of diagnosis. *a.* This has already been given to some extent (¶ 89) in speaking of hypertrophy.

95. *a.* The pulse is weak and irregular in force and rhythm; rarely intermittent. *b.* Apex beat indistinct and not lowered; percussion dullness increased vertically more than transversely; impulse undulatory; ribs not bulged, unless there is also hypertrophy; systolic sound, shorter, clearer and more superficial than in health; defective nutrition; dyspepsia more or less marked. dropsy, especially of lower extremities; tendency to emaciation; cold extremities; feeling of being chilly from defective circulation; palpitation on the least excitement; great præcordial uneasiness; ringing of the ears, headache, indisposition to any labor, mental or bodily; somnolence, in the advanced stages of the disease; evidence of congested liver and kidneys; sexual inclination weakened; superficial pulses not visible; symptoms of bronchitis and pulmonary œdema; in the last stages of the disease, orthopnœa complete; asthmatic paroxysms; occasional respiratory forgetfulness, a fatal symptom; frequent nausea and anorexia; livid discoloration in spots; occasional sphacelus of the finger tips or toes, from capillary stasis; leaden, almost black lips, particularly in the morning.

96. Some or all of these symptoms may be present in a single case. *a.* When but a few of them are present, even the auscultatory and per-

cussion signs only, the diagnosis is established.

97. The differential diagnosis of hypertrophy and dilatation is obvious from what has been already said.

98. As to the *treatment*, it is radically different from hypertrophy. *a.* The treatment is eminently sustaining, directed to the improvement of the quality of the blood, without diminution of its quantity even, for it is not likely to be in excess.

99. Solution of perchloride of iron, largely diluted, is here of special service as a diuretic and tonic. *a.* Opium and quinine combined are constantly required, not more than two or three grains of quinine a day is requisite. It may be given in three or four pills, one at a time, each pill to contain from an eighth to half a grain of opium.

b. If the pulse be rapid, above 90, digitalis may be given in syrup of wild cherry, in half drop doses hourly, until the pulse fall to 85, but it must be given with greater caution than in any affection of the heart, except fatty degeneration.

c. The bowels may be moved daily with any mild aperient; perhaps formula *a*, page 59, will be found convenient. An aloetic pill daily at bedtime, or a castor-oil capsule may answer as well.

d. Taraxacum and calomel are recommended for the same purpose, and are often indicated.

e. Dry cups to the heart in case of extreme palpitation or *angina* often afford instant and enduring relief. *f.* Blood must rarely be taken. *g.* Passive exercise is of great utility. *h.* The patient must lead a cheerful life, and not be exposed to annoyances and hardships; voluntary exercise, such as walking or horseback riding must not be tolerated, except with great caution. No violent emotion or passion should be indulged. ("rupture of a dilated heart has occurred *in actu coitus.*") Animal diet is preferable; much drink of any kind, even if it be milk, is to be avoided; the diet should be rather dry than the contrary, but well prepared and easily digestible. Cold sponge baths may be used, if they agree with the patient, not otherwise. Perhaps tepid or hot salt water bathing is on the whole the best. The clothing, especially of the extremities, should be warm and light; intercurrent diseases must be treated on general principles modified to suit the actual condition of the heart.

100. Ammonia and lavender will afford speedy relief to depressed nervous sensations, and præcordial faintness. *a.* In the latter stages, the patient should be always watched in sleep, on

account of the strong tendency to respiratory forgetfulness, by which life might be suddenly arrested, long before the *necessary* fatal issue of the disease. I will close the consideration of dilatation by a condensed abstract of a peculiar mode of treatment suggested by Piorry, of Paris, in 1858.

101. By a series of observations, carefully made, with the aid of the plessimeter, Piorry satisfied himself that the *heart* and *liver* of a person in health diminish in bulk by forced inspirations. He next undertook to apply this discovery to the diagnosis and treatment of enlarged *liver* and *heart*. I have not attempted to repeat his experiments, but they seem rational, and the opinion of M. Piorry, who has applied himself to percussion and auscultation for now nearly forty years, will hardly be contradicted. But to the facts.

102. M. Piorry selected a hysterical patient, who had nothing the matter with either the heart or liver; he limited the heart by percussion, and found it occupied a space of 11 centimeters * from right to left; the liver occupied a space of 14 centimeters, from above downward. (I omit the other measurements.) This done, he directed

* See note page 75.

the patient to breathe forcibly twenty times in succession, and then repercussed: instead of 11 he found 9 to $9\frac{1}{2}$ for the heart; and instead of 14, about $12\frac{1}{2}$ for the liver. This is not all. He now directed the patient to hold her breath, whereupon he demonstrated that the two organs named not only returned to their natural dimensions, but that they slightly increased in bulk.

103. Now, as this experiment has had the same result on all patients, it is easy to see its influence upon the diagnosis and treatment of diseases of these two organs.

a. What is said above, is, moreover, in complete accord with what is daily observed in pathological anatomy. Uniformly, when a person dies of asphyxia from mucous engorgement of the bronchi, the heart and liver are found to be much enlarged and gorged with blood; whereas, on the contrary, when the respiration is unobstructed up to the last moment of life, these organs are found to be small and bloodless.

104. The practical applications of this phenomenon are the following: It aids in establishing the *diagnosis: a.* Of hypertrophy and dilatation of the heart; *b.* Of hepatemia (congestion of the liver), together with lesions of nutrition of this organ.

105. It aids in determining the rational treatment of dilatation and congestion of the heart, and of some of the *organopathic* conditions which are the consequences of these diseases.

106. *Diagnosis of dilatation and of hypertrophy of the heart.* Hitherto we have had but little more than the following symptoms by which to distinguish *dilatation* from *hypertrophy* of the heart: more extensive dullness at the præcordial region, heart beats feeble, absence of impulse; if these signs were perfectly well marked, we could establish the diagnosis of *dilatation* beyond question; but in the majority of cases there was room for doubt of the exactness of the diagnosis; but by the aid of the physical signs I am about to describe, the diagonsis cannot for a moment remain doubtful.

107. Let us suppose two well-marked cases, one of *hypertrophy*, the other of *dilatation* of the heart. Let us now limit the volume of the heart very accurately, by the aid of percussion; now let us cause the patient to breathe forcibly several times in succession; let us uow measure the heart again with the same accuracy; we shall find in the case of hypertrophy that the heart has undergone no change; whilst in the case of dilatation, we shall find that the heart has returned to about

its normal volume, (that is, to a transverse measurement of about 11 centimeters.)

108. If it be a case of partial *dilatation*, or a partial *hypertrophy* of the cavities of the heart, only the dilated cavity will return to its normal state, while the hypertrophied ventricle will remain unaltered by the forced respirations.

109. If, as most frequently happens, the dilatation be accompanied with hypertrophy, the *diagnosis* will be still more easy, because in such case, the only slight diminution of the increased volume of the heart, will indicate dilatation with hypertrophy.

110. In dilatation, the heart diminishes, by emptying itself of the blood which it contains. A long time ago, Piorry observed that after bleeding a patient with dilated heart, this organ was found diminished in bulk.

111. The application of this same principle to *diagnosis* of congestions, or permanent alterations in the volume of the liver is at once obvious.

112. *Treatment of dilatation of the heart, and its consequences.* The *treatment* of hypertrophy or dilatation, or the two conjoined, follows directly from the means of *diagnosis*.

113. In these cases, if the patient be made to take deep inspirations from time to time, (the more

frequently the better,) amelioration of the symptoms will very soon become manifest. In most cases, especially in dilatation with hypertrophy, the cure will not be so radical, but the patient will experience great relief, and by the disease being thus kept stationary, existence may be prolonged far beyond the limit it would otherwise only reach. Piorry says he has cured or relieved a great many cases by these extremely simple means.

The resultant dropsies, also, often rapidly disappear under the same treatment. This is equally applicable to similar conditions of the liver, and to the serous infiltrations resulting from obstruction to the portal circulation. It is only by the aid of the plessimeter that these nice variations in the volume of the heart or liver can be made out.

Whatever one may think of this mode of *diagnosis* and *treatment*, it is certainly worthy of trial, especially as it is in the last degree harmless.

CHAPTER IX.

SOFTENING, FATTY METAMORPHOSIS OR DEGENERATION, OR FATTY SUBSTITUTION OF THE HEART. RUPTURE OF THE HEART. CASES.

114. The *diagnosis* of softening of the heart is not easy nor absolute. This affection occurs in consequence of adynamic diseases, typhus, typhoid, diphtheria, variola, scarlatina, etc. ; and it results from diathetic conditions of the blood, in which the quality of the fibrin is deteriorated, or the proportion of the red globules is diminished, or that of the white globules increased, as in cyanosis, purpura, scurvy, leucohæmia, glycohæmia and acute phthisis.

115. The heart softens, at least superficially, in pericarditis, and throughout the thickness of its walls in carditis, in hemorrhagic or serous infiltration, in fatty external deposit it is softened superficially, and in fatty degeneration it is softened in portions of the walls of one or both ventricles or auricles, or throughout its entire substance, ventricles, auricles, valves and investing membrane. The detailed analysis of these conditions does not come within the scope of this epitome.

116. What I wish now to consider, is simple

softening of the muscular substance of the heart, without metamorphosis of its histological elements.

Can this kind of softening be verified clinically and exclusive of softening from fatty infiltration or fatty degeneration? I think this question may be promptly answered in the negative. Nor are the causes of the different varieties of softening so clearly appreciable that we may with great probability infer that, in a given case, the softening is or is not attended with fatty degeneration or infiltration.

Still most careful observers will have a settled opinion in their own minds, based on more or less satisfactory reasons. Indeed, the *diagnosis* of softening, as distinguished from *degeneration*, can be rationally made out, and that too with sufficient nearness to constitute a basis for rational treatment.

a. For example: a young adult of active habits, and accustomed to plain food and to total abstinence from alcoholic drinks, is attacked with typhoid fever, and in the course of one, two or three weeks presents the symptoms of softening, viz.: feeble, irregular pulse; much diminished or absent first sound of the heart (there being no mitral regurgitation); heart's impulse invisible

at times, and when visible unaltered in location; impulse, also, but slightly appreciable to the touch, or entirely impalpable; the extremities presenting a leaden hue (all valvular diseases susceptible of exclusion); suppose this case to have præcordial uneasiness, with occasional transient pains in the vicinity of the heart; would it not be extremely probable that he had simple *softening* without *degeneration* or even *dilatation?* Yet the symptoms and physical signs are undistinguishable from those of degeneration.

117. In such case, if the diagnosis of simple softening were correct, the patient would in all probability recover within a period of from six to twelve months, under the use of tonics, nutritious diet, moderate exercise, both passive and active, dry frictions, and cold or tepid sponge-baths, morning or evening, as should best agree with the particular case. Such a case might be allowed moderate doses of whiskey or brandy, or half an ounce, three times a day, of a mixture composed of equal parts of cod-liver oil, whiskey, and syrup of wild cherry bark, or perhaps, better still, Guffroy's cod-liver extract.

118. Is it not possible that the cases of softening of the heart (supposed fatty) mentioned by

Markham (London, 1860,) as having been benefitted by "cod-liver oil in combination with steel," were subjects of *softening* without degeneration?

119. However this may be, we would hardly on theoretical grounds give cod-liver oil to a case with fatty heart; and we should be extremely cautious in the use of stimulants; while, in a case of this kind, both would be indicated in reasonably large doses. As intimated above, (115) the *prognosis* in softening—non-fatty—is very favorable, and would suggest an early and complete recovery.

120. The *diagnosis of fatty degeneration*, though never absolute and exclusive, is made out with so much rationality as to be practically but little less than demonstration.

The *diagnosis* is based on the symptoms already mentioned (115 *b*,) to which may be added defective memory of recent events, inability to perform mental or bodily labor for any considerable length of time; in bad cases, the pulse less frequent than the systole of the heart. Respiratory forgetfulness is often observed; dyspnœa is not unfrequent; and whenever this occurs, the liver habitually congested, rapidly enlarges; relative increased sharpness to second sound of heart.

121. The *arcus senilis* has no value as a symptom of this disease, except in so far as it is in harmony with the general leaden hue of the nails, lips, ears, etc.

122. Syncope, and loss of consciousness *without* fainting, are likely to occur several times in the slow course of this disease; for it is eminently a chronic affection, lasting from a few years to the number of years of human life.

123. Its favorite subjects are persons of luxurious and epicurean habits. Persons who take no exercise and drink freely, are especially liable to this form of softening.

124. CASE.—G. N., of San Francisco, height 5 feet 10 inches, corpulent, weight 170lbs., single, living in perfect idleness on ample income, a very free-liver, aged 38. Drank more than usual on Feb. 7th, 1867, his last birth-day; in the evening fell in a fit. On coming out of this, a few hours after he fell again, but quickly recovered, having sustained no injury but a cut in the skin of the occiput. Fits epilepti-form.

125. He took 30 grains of bromide of potassium, repeated every six hours; had several good naps during the night, and seemed much better on the morning of the 8th. Before night signs of delirium supervened. He talked clearly of all mat-

ters of his past life, told anecdotes with clearness and fluency, but could not remember anything then occurring, from one minute to another ; did not recollect whether he had eaten or drank, or taken the medicine ordered. At 11 A. M., 8th, no pulse except at intervals, then exceedingly weak ; heart beats 150 per minute ; respiration varying from 50 to 80 in three successive minutes ; delirium very mild and subjective ; heart's impulse feeble. The apex strikes the thorax in the fifth interspace, directly under the left nipple. No murmurs on auscultation ; first sound of the heart exceedingly feeble, as if it were an attempt at a contraction which it was unable to complete. The second sound was also muffled, or defective in accent. Respiratory murmur good, except where the left lung was pushed upwards, and condensed by the enormous liver. My *diagnosis* was fatty degeneration, with dilatation; the epileptiform fits resulting from defective supply of arterial blood to the brain, in consequence of heart disease; also, mild delirium tremens.

9th. 11 A. M. Heart beats 140 ; no pulse at wrist ; sweat drenching in abundance, but *not viscid nor cold.* Has persistent clonic spasms, particularly of fingers and wrist joints. He lies flat on his back, and will have no pillow except

the corner of one to raise his head to the level of his body. Bromide of potassium, digitalis, whiskey and beef juice fail to produce any amelioration; by the advice of his consulting physician, he is allowed to have laudanum in such doses as are usually given in delirium tremens, which the consulting physician believed he had, pure and simple. At the fifth dose, given half-hourly, he was asleep; heart beats 140, no pulse; respiration 32; eyes closed, sleep profound, some stertor, clonic spasms of mouth and jaw, pupils not larger in the shade than the head of a pin. One hour later, that is, the 9th, at 10 P. M. he died without having awoke. His respiration was the same, 32, and his heart beats 140 up to the instant of death. His heart stopped, not gradually but abruptly and finally at the same instant. There was no final convulsion.

126. *Post mortem* fourteen hours after, in the presence of several distinguished physicians. There was found *fatty degeneration of heart* and liver. Heart increased about one-third in size. There was no sign of inflammation in or out of the heart; no dropsy; no clots in the heart or vessels; heart pale and flabby; both sets of valves examined with care and found perfect; liver extended above the third rib, was extremely friable and pale. The heart was very soft, being

easily rubbed to a pulp between the thumb and finger ; on section, it presented pale fawn-colored spots on the cut surfaces of both ventricles; these were more distinct than on the surfaces of the heart before being cut. Examination under the microscope showed abundance of oil-globules and amorphous pale muscular substance, but few striated fibrillæ. Externally the heart was not encumbered with more than the usual amount of fat, and this was principally about the base of the right ventricle.

This man never knew he had any disease of the heart, but had complained of oppression about the præcordia before the 7th Feb., and had told a friend he was going to have his heart examined some day, soon. An opiate in large doses is the very worst drug that can be given in degeneration. In this case it is not probable that it hastened the fatal result, for the case would have died in a few hours more, in convulsions, had the nervous system been left to itself; but it is observed that it diminished respiration (from an average of 65 to 32) without affecting the heart beats, which was equivalent to increasing the dyspnœa ; the dyspnœa increased the congestion of the liver and the engorgement of the heart's cavities.

127. Case of Rupture of the Heart; Fatty Softening; Dilatation.—Q., *died suddenly*. The autopsy revealed the following facts: The beginning of aorta was atheromatous; the right coronary *sinus* was converted into an aneurismal sac the size of a hen's egg. The coats of the artery were so far ruptured on the anterior part of the sac, that ecchymosis was visible under the external serous covering. This rupture was incomplete, and about the size of the little finger nail. There was also rupture of the apex of left ventricle, through the ventricular wall which at this point was not a line in thickness. The rupture was incomplete, like that of the aneurismal sac, extending only to the viceral layer of pericardium, which was glossy, and exhibited no sign of inflammation. No blood had escaped, either from the aneurismal sac, or the apex of ventricle under the pericardium. There was a faint attempt at reparation in both places, shown by the deposition of *laminæ* of fibrin, but this attempt had evidently been quite abortive; the deposited laminæ were almost black, and dirty looking. The place of rupture of the ventricle was indicated externally by the ecchymosis seen through the pericardium. The heart was full twice the normal size, very soft, and evidently degenerated, by the substitution of fat for sarcous elements.

Mitral valves incompetent; aortic valves perfect; the other valves were not examined. The subject was a quadroon, aged 30, fine-looking, not emaciated. The pleural sac and peritoneum were distended with effusion; the liver was much enlarged, the lungs healthy. There were several chafes of pleuritis, with deposition of lymph, on the mural layer of pleura, near the apex of the left lung, posterior aspect.

128. This was a case of sudden death. The *post mortem* reveals the proximate cause to have been rupture of the left ventricle. The rupture occurred in consequence of dilatation, conjoined with degeneration of structure elements. The aneurismal incomplete rupture was also much aided by the fatty degeneration of the aortic walls.

129. Violent traumatic rupture of the heart, even when the laceration is very extensive, does not necessarily cause *instant* death, not even fainting. Morel-la-Vallee saw a case in detail at the hospital of St. Anthony, Paris, in 1858. A man of sixty was thrown from an omnibus, a wheel of which ran over his chest, crushing it in a frightful manner. He was immediately carried into the hospital in front of which the accident occurred. Although in a dying condition, he

made some signs to his wife, who was with him, breathed a few times, but was pulseless, and expired. On examination, the left ventricle was lacerated the whole length of its anterior aspect; the upper part of the septum of the ventricles and the left auricle were also torn completely through; the mitral valve was torn in pieces, the tendinous cords were torn out, and remained hanging to the shreds of a fleshy column.

130. The injury to the heart appeared to result from excessive pressure. Although the sternum and ribs were much fractured, no osseous fragment corresponded with any rent of the heart.

131. This case, though not properly within the scope of my subject, is interesting in a physiological point of view, as showing what extensive injury the heart itself may sustain without causing instantaneous death, or even loss of consciousness. But to return to my subject—*fatty heart*—is recognized with great difficulty; but the most reliable negative symptom, is the absence of dropsy. The most reliable positive symptoms are, liability to "heart faintness" and intermittent pulse, while the systoles of the ventricles are regular and *not* intermittent. In the advanced stage of fatty degeneration—and this is the stage in which our attention is most frequently called to it—this

symptom of pulse weak or absent, out of proportion to the systoles, is nearly always present. There is another marked symptom in this stage; that is, a muffled toneless first sound, quite distinguishable from the *accented* sound from dilatation without degeneration, and also, relatively higher toned and more accented *second* sound. Precordial uneasiness or distress is also a marked and frequent symptom.

132. *Summary of Treatment.*—There is scarcely a doubt that fatty degeneration may be arrested, and that the unaffected portions of the heart may be so far fortified as to perform the work of a sound organ, so that its deficiencies will cause no suffering, or even uneasiness, to the subject. This amelioration can be brought about by hygienic and medicinal means in many cases.

133. The hygienic means are frequent bathing —twice a week—pure air night and day; uniform temperature of the surface of the body, by light, warm clothing, passive exercise in salubrious districts of country; such as carriage, boat and horseback riding; little or no benefit from walking need be expected. Food should be nutritious and easily digested. Fish, meat, game, and a rather limited amount of vegetable food may be allowed. Less fatty, starchy or saccharine matter should enter into the diet than in other diseases,

except there be good reason to believe that the softening of the heart is non-fatty. All alcoholic beverages should be avoided, except for the immediate feeling of faintness. All sudden excitements, or *causes of emotions*, should be avoided with great care.

134. The most reliable medicinal remedies are solution of perchloride of iron, largely diluted—a drop to a drachm of water—strynchia, brucia, arsenic. * Quinine and opium are not to be forgotten (see page 63, *c.*); aloetic pills, or some equivalent, are required from time to time, or formula *a*, (page 57,) may be employed with advantage. Electro-magnetism, of very low tension, applied over the pneumogastric nerves, or over the heart itself is worthy of trial. I have no experience of its effects in these cases, but theoretically believe it must be beneficial, if applied once or twice a day, with great caution, for many months. The use of it for a few times could have no result.

135. Finally: fatty heart is very frequent in this city, and has proved fatal in many instances

* Of all the preparations of arsenic, that first recommended by Athar Ali Khan, of Delhi, is most used by me. R. White arsenic, grains 2; black-pepper, grains 100. M. Make, according to art, into sixty pills. S. Dose, one a day for a week, and finally three a day indefinitely.

within my own observation. It favors the formation of clots in the heart—a cause of sudden death. In almost all cases of rupture of the heart, without external violence, fatty degeneration is the predisposing cause—all cases of rupture die within a few moments.

136. It is one of the diseases of the heart that may be treated with the most rational hopes of amelioration and indefinite prolongation of life, if recognized and treated before it has become complicated with *excessive* dilatation, or before it has reached that stage in which the pulse is irregular, very weak, tremulous and intermittent at the wrist, before fainting fits are frequent; in short, before the greater, instead of the less portion of the heart has undergone fatty substitution. In this last stage, any treatment—the most rational—can barely alleviate temporarily, without the slightest hope of retarding the onward progress of the disease.

137. Angina Pectoris, or "suffocative breast pang," is a disease from which the poor are usually exempt. It attacks about nine males to one female. It scarcely ever occurs before the fortieth year, seldom before the fiftieth. It is usually protracted in its course. Death has been known to follow within two hours of the first

attack in a patient aged 74. (Latham.) The average duration is unknown.

138. No disease of the heart is more certainly recognizable than angina pectoris. It consists of spasm and pain in the breast, generally about the lower portion of the sternum, but sometimes as high as the upper end of this bone. The pain runs through the breast to the back, and down the left arm. There is a sense of suffocation, and a feeling that death is imminent. This feeling of speedy dissolution is never wanting, any more than the pain, the suffocation and the constriction about the heart. It comes suddenly, without warning, and goes suddenly and completely. A patient of mine was walking in the street; all at once he dropped down on the pavement, with intense tearing pain, and a feeling of suffocation and instant death. He immediately arose, before two gentlemen near him, who sprang to his assistance had time to reach him. For an instant, he says, he must have been unconscious, for he does not remember falling, but was conscious while getting up. The gentlemen who wished to help him asked how he came to fall so suddenly and get up instantly. He answered that he did not know, and asked how long he had been down? They replied, "No time at

all; you got up as soon as you fell, before we could get to you." This man has had no attack since, but by my advice he has arranged his affairs. A paroxysm of angina pectoris may last several minutes; that this was a case in which material unsoundness of the heart, its valves and of the thoracic aorta were present is indubitable, from examinations carefully made, previous and subsequent to his fall.

139. In order to appreciate the manner of *diagnosis*, it is proper to glance at the usual pathological condition of the heart in fatal cases.

a. In almost all cases, the heart walls, especially of the right ventricle are thinned. The heart is usually softened, generally by fatty metamorphosis. Neither clots in the heart nor emboli in the arteries have been found. The corronary arteries are frequently more or less ossified or constricted by atheromatous deposits. *No matter what the cadaveric rigidity may be, the heart will always be found soft.*

140. The pain of angina is neuralgic, not that of tenderness. It depends, then, on lack of supply of red blood to the heart itself, or upon impaired nutrition or irritation of that branch of the *vagus* which supplies the heart.

Any cause which will have the effect of tem-

porarily diminishing the supply of arterial blood to, or retard the return of venous blood from the heart; or any cause which shall impair the normal function of the vagus may produce a paroxysm of *angina pectoris*. No causes could be more likely to induce such defect of blood supply to the heart than fatty metamorphosis, or dilated heart, or narrowed corronary arteries—the three precise conditions in which we find angina most likely to occur. Here, for the hundredth time, theory and fact coincide.

141. We may expect in most cases of angina pectoris a feeble, small and over frequent pulse, as in dilatation; but from the pulse alone nothing can be determined in this affection. The *diagnosis* rests entirely on the character—the unmistakable character of the paroxysm—great dread of death, a feeling of inability to breathe, for fear of hurting the heart, or causing death, more than a consciousness of real inability to take a full inspiration.

The Treatment of the attack itself is not often committed to the physician, because it is over before he arrives; but in protracted paroxysms, or in those of frequent recurrence in the same day he may be present.

142. Dry cups to the præcordia afford the most

prompt relief. Stimulants, in small, often repeated doses, should be given at the same time, if the patient can swallow. A touch of the cautery between the shoulders, will undo the spasm of the heart most promptly. If the stomach is full, it should be relieved by a prompt emetic —sulphate of zinc or powdered ipecac, a large dose in a goblet of tepid water. If the patient be of full habit, and if it be known that the heart is not dilated, and if serious fatty degeneration be not suspected, and if the pulse be nearly natural in size and fullness and not slow, bleeding at the arm is certainly indicated, or blood may be taken by scarification and cups from the præcordia, if not thought proper to risk the opening of a vein.

143. But the most important part of the treatment of a subject of angina pectoris is that directed to the postponing of the recurrence of the attack, which is sure to come again, sooner or later, and with more violence; and the more frequently the greater the number of paroxysms already endured. All emotional influences must be avoided. The temper must be controlled. The passions indulged with the greatest moderation. The natural calls of the body obeyed with deliberation; hence the bowels must not become

costive, nor the bladder distended. There must be no violent or forced work, or exercise, or amusement. No severe or rapid intellectual labor; no excesses of any kind—moral, æsthetic, physical, mental or emotional. Daily passive exercise in the open air; slow walking on level ground in open spaces, as in the country or suburbs, or unfrequented streets, may be permitted.

144. Riding in a carriage is much preferable to riding on horseback, however easy the gait of the animal, because of the labor of mounting and dismounting, and the risk of sudden motions of the horse, which might bring on a fatal paroxysm.

Tonics are generally indicated, also stimulants and nitrogenous nutrients, in rational quantities. If anemia be present, iron must be employed, and cod liver oil, in the modes already stated, as in dilatation, or in softening, without fatty metamorphosis.

145. Dr. WALSHE, with great theoretical propriety, says, "The removal of gout, chronic rheumatism, or old standing skin diseases, should be very cautiously, if at all attempted, in the subject of angina; relief of those complaints is unquestionably sometimes followed by increased severity of the cardiac affection."

146. *Rheumatism*, which occupies so important a place in the consideration of pericarditis and endocarditis, requires mere mention. It is justly dreaded on account of the occult effects it is likely to leave in the organism. Persons who had rheumatism years ago, are constantly seeking medical advice for some trouble about the heart, although at the time of the rheumatism no heart symptoms were observed. Is it not probable that some lesions of the heart, though not derived from rheumatism result from similar diseased action, or from the presence in the blood of similar morbific elements?

147. I will assume that rheumatism is recognizable by all physicians, and will merely allude to what appears to me to be two well-marked varieties of this affection. The one I will call inflammatory rheumatism, which is most to be dreaded, as to its effects on the heart; the other I will call *neuralgic rheumatism*. The former is attended with fever, tenderness and acute pain and immobility of the muscles and joints implicated. The latter is recognized by a persistent dull ache, increased to an ache with partial numbness, by excessive use of the affected limb or part, but never attended with those flashes of agony which characterize pure neuralgia. This

kind of rheumatism is certainly curable in the great majority of cases with quinine and opium in combination, given in small doses, aided by colchicum and whiskey given in regular doses and in mixture. Alcoholic beverages are to be avoided ; vinegar, sugar and fruits must not be used. If there is anemia, cod-liver oil, with syrup of wild cherry and whiskey will hasten the cure. From one to six months of this treatment will be required, in neuralgic rheumatism of the hip joint after gonorrhœa, or in neuralgic rheumatism of the lumbar region, or of the shoulders, or feet and ankles.

148. Acute or chronic inflammatory rheumatism is certainly curable in a great majority of cases by propylamin and laudanum, and aloetic aperients. But I am of the opinion that it will often be found requisite to give ten times the dose recommended by Dr. Awenarius, who first recommended it in this disease. I have cured so many cases with these means alone, that I now use scarcely anything else. I have had no success with stimulants, nor with fatty food. Here, as in neuralgic rheumatism, vinegar, sugar, fruits, fermented beverages, etc., are not tolerated, except there be anemia as a complication.

149. Leeches afford temporary relief, so do

stimulating liniments, and anodyne cataplasms; indeed, moist heat, no matter how applied, so that the air is excluded from the affected part. Cathartics and small bleedings are no doubt often of great benefit in the beginning of the affection, if the patient be of full habit and in good blood-making condition ; but after the first few days, bleeding is worse than useless. The aloetic pills, on the other hand, may be continued during the whole course in laxative doses.

150. Absolute rest, in an apartment of uniform and rather high temperature materially hastens the cure. The diet should consist of rather thin broths, with plenty of salt and no pepper. No other alkali than the propylamin will be needed.

The *sulphate of manganese*, already mentioned, I have no experience in, but it is well worthy of trial as a preventive of the cardiac effects of rheumatism.

CHAPTER X.

THORACIC ANEURISMS: DIAGNOSIS AND TREATMENT. CYNOSIS AND ATELECTASIS.

151. The anatomical relations of the great blood vessels of the chest render the diseases of these structures most dangerous in their tendencies and results.

152. Under the head of thoracic aneurism may be comprehended aneurism of the aorta, or of the innominata, pulmonary or bronchial arteries. The diagnosis of thoracic aneurism in its earlier stages is always difficult and frequently quite impossible; for although its presence is always attended with more or less objective and subjective symptoms, still there is no known pathognomonic indication upon which we can place implicit reliance. As the tumor progresses in growth, and fluctuation and pulsation become evident, of course there can no longer remain any doubt as to the nature of the disease. The diagnosis of aneurism of the aorta will be aided by a consideration of the mechanical effects such an enlargement might be expected to produce, and also the peculiar functional disturbances which would most likely ensue from the consequent pressure upon surrounding and adjacent structures.

153. Thus, if a patient present no satisfactory evidence of a stricture of the œsophagus, and yet dysphagia be present, with an apparent obstruction to the passage of food at a point just opposite the second intercostal space, aneurism of the arch may be suspected. A similar obstruction in the œsophagus at a lower point might be owing to enlargement of the descending aorta, and if still lower and just above the diaphragm, aneurism of the thoracic aorta where this vessel is crossed by the œsophagus would be probable. The attendant functional derangements vary with the location of the aneurism. Thus, spasmodic laryngeal affections would indicate pressure upon the recurrent branch of the pneumogastric nerve, at a point corresponding with the posterior and inferior portion of the arch of the aorta, and hence be suggestive of aneurism at this point, or of the arteria innominata. If constriction of the chest be a prominent symptom, aneurism of the ascending portion of the arch, with consequent pressure upon the cardiac plexus would be probable ; if orthopnœa or asthma, the commencement of the descending portion, etc.

154. Displacement of the heart, not otherwise accounted for—weak systole—contraction of the pupils from pressure upon the sympathetic nerve, evidence of aneurismal diathesis, hæmoptysis,

angina pectoris, raucous voice, aphonia, intercostal neuralgia; pain radiating from the chest, or fixed at the upper portion of the thorax, swelling of the feet or infiltrated condition of the eyelids are signs which will be construed by the intelligent physician, as their import in any given case may justify.

155. As the tumor enlarges, it may project above the sternum, or by its pressure produce absorption of the osseous and cartilaginous walls of the chest, presenting itself as a soft, palpable and pulsating tumor. The above remarks are all, more or less, applicable to aneurism of either the pulmonary arteries or of the innominata; the peculiar modifications dependent upon the anatomical relations of these vessels being remembered. Thus, in aneurism of the innominata, the tumor can be felt and its boundaries defined; it may also produce dyspnœa and dysphagia, by its pressure upon the nerves concerned in respiration and deglutition. Its pressure upon the subclavian artery may obliterate the pulse at the right wrist; or, if upon the trachea, threaten suffocation. A full consideration of the lesions of this short but important artery belongs more properly to the province of surgery than to that of practical medicine.

156. Aneurisms of the pulmonary or bronchial arteries are comparatively rare, and their diagnosis is, perhaps, in most cases impracticable. We should here, however, expect the symptoms to be more intimately connected with pulmonary complications than in similar affections of the aorta or innominata. In such obscure cases the practitioner should be guided by the light of anatomy and physiology, without which the most simple problem in diagnosis may become an incomprehensible mystery.

157. Auscultation and percussion are invaluable as enabling us in many instances to diagnose thoracic aneurisms before their presence could be communicated to the senses through any other channel; they are neither, however, infallible, and as is true of all other means at our command, are only susceptible of a limited practical application.

158. The "aneurismal bruit" is neither constant nor pathognomonic, but is of value when taken in connection with other signs.

159. When this *bruit* is present, it is not unlike the sound produced by whispering the letters h-w-h-e-e-u-w, thrown together in the form of a word; it may, however, vary in pitch and intensity, be slightly musical, rasping or blowing, or

all these variously combined. A "jogging" sound is supposed to indicate aortic dilatation. When these sounds are heard, they are, of course, systolic, and must be distinguished from valvular murmurs by their location, direction of propagation, etc. Percussion may be of value. It can prove that resonance is decreased over the region of the disease in these cases; it tells us nothing more, however, except the probable size of the tumor. It does not tell us whether the dullness is caused by a fibrous, encephaloid or aneurismal growth.

160. *Treatment.*—The disease is incurable; and if we can prevent, temporarily the fatal issue, it is all that can be expected. Spontaneous cure is a theoretical possibility, but too remote to constitute a foundation for reasonable hope. The treatment should consist of such management of the case as will be most likely to keep the circulation equalized, and in theory better still if the heart's action is constantly kept below its normal force. The habits of the patient should be strictly regular and temperate in all things. Venereal excesses should be especially forbidden, and passive rather than active exercise permitted. In some advanced cases, absolute rest must be enjoined. Tracheotomy may temporarily prolong life where laryngeal symptoms threaten imme-

diate suffocation. The treatment of aneurisms of the innominata is more properly a surgical topic. The innominata has been ligated a number of times, but only once successfully.

161. *Cyanosis* is generally the result of a congenital defect in the heart, such as the non-closure of the foramen ovale, and of the ductus-arteriosus. There may be a congenital opening in the septum ventriculorum. It may also depend on arrest of growth, either before or after birth, of the root of the pulmonary artery, whereby its orifice is rendered permanently too small; this may be the only congenital defect, the venous and arterial blood not mixing at all. In this case, the blue tinge of the skin is as easily accounted for as in the other malformations.

Rokitansky has seen cases of narrowed pulmonary artery without cyanosis. This fact has been often remarked by other observers. The foramen ovale is frequently open, and yet there is no commingling of arterial and venous blood, unless the pulmonary orifice is constricted. Hence it is probable that obstruction at the *ostia* of the heart, from constriction or other cause, combined with patent foramen ovale, open septum ventriculorum, patent ductus arteriosis, or insufficient tricuspid valves, is necessary to the production

of cyanosis. The venous stasis, and red and purple blood mixture, theories are neither true alone, but when both exist at the same time, *cyanosis* must result. But really the consideration of this disease hardly comes within my theme—it belongs to works on malformations of the heart, among which that of Dr. Peacock is excellent; but I will merely add, that we shall find hypertrophy of the right ventricle, etc., etc., the same catalogue as that which results from mitral obstruction and patency. The treatment is not peculiar, but more difficult, because it has to do with children who have not yet arrived at accountable age. Passive exercise, slow movements, restricted and select diet, pure air, special treatment on reflex principles, of the paroxysms of dyspnœa or syncope that frequently occur, are all that can be suggested. The affection is in the end fatal, and this result may be postponed, but not averted, even by the most rational treatment.

162. The lividity of *atelectasis* of the lungs, (non-distension of air vesicles,) sometimes manifested in the new-born, cannot be mistaken for cyanosis, resulting from malformed heart. The former sometimes disappears after a violent fit of coughing; the latter is persistent during life.

CHAPTER XI.

General Rules for Diagnosis of Diseases of the Heart.

I. If the patient, otherwise in good health, complain of uneasiness in the præcordial region, cardiac disease may be expected.

II. If a murmur be heard at the base systolic in time, (that is, with the first sound of the heart) which diminishes in intensity as the ear is moved towards the left nipple, it indicates roughness of the semilunar valves, or constriction or roughness of the aortic orifice.

III. If a systolic murmur be heard at the præcordia, and if it increase in intensity as the ear is moved toward the left nipple, and diminish as the ear is moved up the sternum, it indicates *mitral regurgitation*, that is, insufficiency of the mitral valves.

IV. A murmur heard at the base of the heart, (about the junction of the third ribs with the sternum,) thence down the sternum, coincident with the *diastole*, (second sound of the heart,) indicates *aortic regurgitation*. If to this sign be

added visible superficial pulses, and a hammering pulse at the wrist, aortic regurgitation is certain.

V. A murmur coincident with the second sound heard about the left nipple, or in the fifth interspace below and to the right of left nipple, and along the heart towards its base, and no where else, indicates mitral constriction; [this murmur is very rarely heard.] If to this be added a slight cough, the lungs being sound, and hypertrophy of the right ventricle—mitral constriction is indubitable.

VI. A murmur heard loudest above the base of the heart in the upper part of the thorax, indicates aneurism of the aorta, or innominata, or subclavian artery. If to this sign be added a pulse of unequal strength in the two wrists, or absent in one wrist, aneurism is almost certain. Difficulty of deglutition and paroxsyms of dyspnœa add greatly to the probabilities of aneurism; indeed, with the preceding signs, render it indubitable.

VII. If there be bulging of the left side, near the mid.sternum, and heaving impulse of the heart, and strong full pulse, there is *hypertrophy* of the heart.

VIII. If there be a visible undulatory impulse, no heaving of chest if the pulse be not strong

nor very resistant, if the first sound of the heart be clear and more distinct, and seem nearer the ear, and have more of a knocking character, than normal, there is *dilatation* of the heart. If there be much bulging of the interspaces, and if the pulse be strong for dilatation and not strong enough for hypertrophy; if the apex be outside of the left nipple and below the sixth rib, there is hypertrophy with dilatation. If there be also dropsy of the lower extremities, the probabilities of dilatation become certainties.

IX. Basic murmur, coincident with first sound, heard loudest at the junction of the third costal cartilage with the sternum, and thence down the sternum, attended with persistent jugular pulse, indicates almost positively tricuspid regurgitation—insufficiency of the tricuspid valves—if to these two signs general turgidity of the venous system be added *tricuspid regurgitation* becomes certain.

X. An *endocardial murmur*, whether systolic or diastolic, whether at the base or apex, heard suddenly during the course of an acute rheumatism, or after a violent blow on the præcordia, or during Bright's disease of the kidneys, indicates *endocarditis* in the most positive manner.

XI. An *attrition* or *friction* sound heard over

the præcordia, (¶ 5, p. 11,) that is, over the fifth left costal cartilage, while the patient holds his breath, indicates *pericarditis*, in the most positive manner.

XII. *Softening of the heart*, without fatty degeneration, occurs only in cases of asthenic or adynamic diseases of an inflammatory nature. If in such cases the pulse grow feeble out of ratio, with the intensity of the adynamic disease—for example, a typhus or typhoid fever—and remain weak and unequal, become easily excited and fluttering, if at the same time the patient feel steady præcordial uneasiness, *softening*, of the non-fatty variety, is extremely probable.

XIII. If the same symptoms mentioned in the foregoing rule be observed in a *bon-vivant* of luxurious and idle habits, especially if he be at the same time an intemperate drinker, *fatty degeneration* of the heart is almost indubitable. If to these symptoms be added epileptiform seizures, and if the respiration varies greatly in uniformity as to frequency and force without any external cause, and at the same time the patient be over forty years old, *fatty degeneration* may be considered certain.

XIV. *Angina pectoris* cannot be mistaken if

the suffocation præcordial pain, the dread of imminent death, have once occurred, so as to be described by the patient in these or similar terms, without having been questioned by the physician. The symptoms pertain, in their clearness and pertinence, to no other affection.

XV. A *murmur* coincident with the first sound heard at the base and propagated up the aorta, in an anemic person, or in a person whose blood under the microscope exhibits defect of red or excess of white globules, is an inorganic *murmur*, and indicates merely altered condition of the blood, or altered dynamism of the heart.

General Principles of Treatment of Diseases of the Heart.

I. The first and most important principle to be kept in view in treating a diseased heart is *to diminish the labor it has to perform.*

II. This is done in two ways. *a.* Directly, by diminishing the amount of blood in the body; *b.* By *diminishing* the functional activity of all the organs not directly concerned in secretion, and by *increasing* the functional activity of the skin, liver, kidneys, lungs and alimentary canal.

III. *a.* Blood may be abstracted directly, either from the arm by venesection, or from the præcordia by cups, when from general plethora or overwhelming local congestion, a sudden diversion to the blood current is deemed essential; or the total quantity of blood in the system may be diminished by reducing the weight of the body—the loss of eight pounds of weight is the loss of one pound of blood.

b. Blood never is to be taken with a view of cutting short an inflammation, or *curing* either an acute or chronic affection, but merely for the purpose of relieving an urgent symptom, or arresting an imminent catastrophe.

c. On the other hand, bleeding is not so hazardous as many would wish us to believe. *d.* If the digestive organs are unimpaired, loss of blood by hemorrhage is restored with astonishing rapidity. *e.* In this respect, anemia from traumatic hemorrhage or venesection is widely different from pathological anemia, depending on lesions of nutrition, assimilation and innervation; in short, on lesions of all the organs of the body. *f.* Blood deterioration in this case is repaired with extreme slowness, and this is not to be used as an argument against venesection, leeching, or cupping. A few pints of beef tea will restore as

many red globules as are removed by a copious bleeding.

IV. So local bleeding, even in *anemia* of slow growth and long duration, is not always objectionable to mitigate a threatening local symptom. *a.* For example: take a case in another department of pathology. A child of six months, more or less, badly nourished with insufficient mother's milk, and cow's milk and farinaceous mixtures, has chronic diarrhœa. In the course of treatment we intentionally diminish without arresting the diarrhœa: the child now has tonic spasm of the fingers and toes, the eyes pitch back under the brow, or look straight onward, seeing nothing; it rolls its head and moans, and starts with sudden shrieks. This child is anemic, very pale, waxy almost, but its head is burning hot. Now, if a single leech be applied at the anterior fontanelle and allowed to fill, and after it drops off, the bleeding be encouraged by the application of compresses wrung out of hot water, not only over the bite, but over the whole top of the head, for the space of a whole hour; if this be done within the first twelve hours after the tonic spasm becomes manifest, the chances are more than two in three the child will recover, providing it be nourished with *beef juice*, and medicated solely

with minute doses of creosote subsequently, and kept warm at the abdomen and extremities. *b.* Now, this is a case in which an apology is needed for bleeding, if an apology is ever needed; yet I have bled in twenty recorded cases in this manner, and in seventeen have had the satisfaction of seeing my little patients recover.

V. As a general rule, *depletion* by bleeding is not required in diseases of the heart.

VI. By abstinence from hard labor, mental or physical; by abstinence from all severe exercise, and by avoiding all violent emotions or passions, the demands of the heart are obviously lessened, and hence its labor lightened.

VII. By aperients regularly taken, by diuretics often repeated, by hepatic stimulants taken from time to time, by unobstructed access to the atmosphere at all hours of the day and night, by baths, frictions and abundant passive exercise, the functions of the liver, skin, lungs, kidneys and alimentary canal are certainly increased, and that not in a way to be detrimental to alimentation, assimilation and nutrition.

VIII. Whatever degree of sedative impression we desire to make on the heart, must be made slowly, not suddenly. A violent blow on the pit of the stomach will stop the heart forever. A

thousand light blows or a heavy weight would produce but little effect. Small doses of cardiac sedatives, often repeated and carefully watched, are better than large ones, however judiciously administered.

IX. *The quality of the blood must be maintained as near the normal standard as possible, while its quantity may be diminished by restricted diet, and certain evacuants, in proportion as the disease of the heart advances.*

CHAPTER XII.

GENERAL REFLECTIONS AS TO PROGNOSIS IN ORGANIC DISEASES OF THE HEART.

163. Heart disease destroys life in three ways: *a*, by *syncope ; b*, by *apnœa ; c*, by so interfering with assimilation as to cause gradual destruction of life from inanition—starvation, not from defective food or defective appetite, but from defective assimilation and æration of the blood.

By far the most common special organic disease of the heart is that of the mitral valves. Insufficiency of the mitral valve is exceedingly common in this city : cases may be counted by hundreds. Now, what is the result? Insufficiency of this

valve is not incompatible with the longest life. If the insufficiency be slight, but little blood is repelled at the ventricular systole into the left auricle, and this becomes slightly or not at all hypertrophied; the left ventricle in such case may remain unhypertrophied. But the usual course is, the left auricle thickens; the pulmonary vessels become congested, more or less, with the refluent blood; this reacts on the right ventricle, which becomes hypertrophied. Here the case may remain stationary for years, or for life; and rational treatment may greatly help to insure this result; but usually, from neglect or otherwise, the disease advances. The thin-walled right ventricle, being only designed for impelling the venous blood through the pulmonary arteries and capillaries around to the left side of the heart, laboring under the great burden of rejecting the increased volume of blood thrown back upon it from the powerful left ventricle, now itself reinforced by hypertrophy, and aided in its damaging ability by the dilated pulmonary vessels, begins to dilate. Now nature steps in again, to counteract one disease by another. The tricuspid valves become incompetent, from the dilatation of the right ventricle, and part of the venous blood is repelled by the systole of the right ventricle upon the *venæ cavæ*, and again

there is rest. Even now, by judicious treatment, by reducing the weight of the body, and hence the volume of blood, while assimilation is not allowed to become impaired, life may continue for many years, and the patient die perhaps of old age. But generally no steady persistent treatment is permitted by the patient. He goes from one good physician to another, and obeys none long enough to receive permanent benefit, and in the nature of the case he is unable to treat himself judiciously; hence the disease enters on its third and last stage. The heart in its enfeebled condition being overworked, by having to impel too much blood relative to its ability, makes new efforts at reparation in detail, only to hasten the ultimate result: that of rendering the heart totally incapable of performing its functions; thus, the left ventricle becomes more hypertrophied, and if the left auricle has not yielded to dilatation in excess of hypertrophy, the left ventricle may also become slightly dilated at first; but this dilatation never advances far, for the left auricle soon yields, and becomes so dilated as no longer to be able to affect the ventricle by its contractions. By this time the dilatation of the *right ventricle* has become enormous, and in excess of its hypertrophy. The tricuspid is now constantly insufficient; the jugular

pulse is always visible, night and day; the lungs are congested; the breath gives out on the least hurry. There is œdema of the lower extremities, dyspepsia, congested liver, still more oppressed respiration, greater lack of æration of blood in the pulmonic vesicles; face looks purple, or deep red, perhaps maroon-colored. The patient is very much disposed to be cold, seldom perspires; there is occasional spitting of blood; the pulse becomes weaker and less uniform, even in its weakness. The end of such a case is obvious; but often such a progress and result might be almost indefinitely postponed. Insufficiency of the mitral valve, while it is the most common, is fortunately the least fatal in its tendency of all the organic diseases of the heart, and the most controllable by systematic treatment.

164. Mitral constriction, if slight, is not incompatible with tolerable health and long life; but when the constriction is considerable, its consequences follow in more rapid succession. Here the left heart remains unaffected, or may become atrophied for *lack* of work; for obviously, if the constriction is so great as to admit but half the blood current in a given time, the left heart has but half work to perform. A moderate constriction nature compensates, by the hypertrophy of

the right ventricle, and probably of the pulmonary arterial walls also. The right ventricle also becomes hypertrophied all the more when the constriction is considerable, and this is, of course, curative in its tendency; and if the hypertrophy of the right ventricle balance the constriction of the mitral orifice, so that by its action the same amount of blood is forced through in a given time, as though it were not constricted, then the hypertrophy is *absolutely* compensatory, and practically the patient is cured. This no doubt does sometimes happen and continue through a long life. But generally it is far otherwise.

165. The blood not being able to flow into the left ventricle in normal volume, accumulates in the left auricle, which hypertrophies by increased function; but still being unable to impel the blood onward as fast as it arrives, part of its contractile force is expended backwards upon the pulmonary veins, which becoming congested, impede the onward current from the pulmonary arteries, and so back to the right heart, as in mitral insufficiency. Insufficiency being almost always joined with constriction, we get the same train of symptoms exaggerated, to which may be added tendency to syncope, and sometimes, though rarely, death from this cause; because enough

blood may not reach the arteries, in consequence of the constriction, to supply the brain ; but almost always death occurs by apnœa, as previously stated.

166. *Disease of aortic valves*, whether insufficiency or constriction, produces an opposite train of symptoms, and is more speedily lethal in its tendency; and, indeed, is less controllable by treatment, whether hygienic or remedial.

167. Aortic obstruction, indicated by a systolic murmur, loudest at the base, that is, about the middle of the sternum, opposite to the third intercostal spaces, and aortic insufficiency, indicated by a diastolic murmur, heard loudest at the same place, and propagated down the sternum, are more commonly met with in middle or advanced age than in earlier life, and more frequently in males than females; while females are more liable to mitral disease than to any other affection of the heart.

168. These two affections of the aortic valves, when of long standing, are generally attended with enormous eccentric hypertrophy of the left ventricle, and final incompetency of the mitral valve, congestion of the pulmonary vessels, and finally, if the patient live long enough, hypertro-

phy and dilatation of the right heart, tricuspid regurgitation, engorged systemic veins, impaired blood, serous infiltrations, dyspnœa, cough, syncope and death. The order mentioned is the usual one in which nature attempts restoration at every step of the advancing disease.

169. Hypertrophy of the left ventricle is compensatory of obstruction or regurgitation at the aortic valves, or of both; dilatation is a new step in the fatal direction, and then mitral regurgitation comes in as compensatory to that. Left auricular hypertrophy and dilatation tends to prevent the excessive injury to the pulmonary vessels, by recoil from the ventricle, and then, when this resource fails, the pulmonic valves increase in strength, and the right ventricle takes on hypertrophy, as a new compensation for the ever widening mitral orifice.

170. Then another step in disease is dilatation of the right ventricle, followed by a compensatory lesion, namely incompetency of the tricuspid valves, with its attendant regurgitation; then disease, in the giving way of the valves of the veins, before the refluent venous current and the attendant venous congestions; then a final and last effort at compensation in serous exosmosis or dropsy.

171. Thus it is seen that each advance of the disease is met by a compensatory lesion, which, while it adds to the sum total of organic damage, retards the fatal progress of each integral lesion, reinforced, as it is, with all its antecedents.

172. At each of these compensatory struggles, art is able to give nature valuable aid in the prolongation of life, and in averting sudden accidents.

173. A small aortic obstruction or a trifling insufficiency of the semilunar valves, may be so accurately compensated for by hypertrophy as to produce inappreciable results, and not noticably shorten life. A considerable insufficiency, without obstruction, may be so compensated by hypertrophy as to produce no evil consequences, so long as the mitral valves remain competent; and this latter result may be indefinitely postponed by such treatment as has been suggested in former chapters.

174. The narrowing of the aortic orifice to one-fifth its normal condition, is not incompatible with the continuation of life and the enjoyment of unsuspected health, providing it be recognized, and the patient placed under rational treatment and constant care; for it is quite probable the left ventricle may become so strengthened

by hypertrophy and innervation, as to be able to propel the blood with a velocity sufficiently increased to compensate for the defective aperture through which it has to pass.

175. Still it is not denied that aortic valvular diseases are generally fatal at no long period after their recognition, much more promptly than other valvular lesions. A little hurry, a violent passion, a sudden motion, is liable to surprise the heart and cause its arrest for an instant, during which syncope takes place, from lack of supply of arterial blood to the ganglia, and this syncope is too often final.

176. *Tricuspid regurgitation* is a fatal affection and only less dangerous as to the imminence of its result than aortic valvular disease. A fatal termination may be a long time postponed, by diminishing the volume of blood in the system, by means of restricted diet, diuretics, evacuants, etc., according to the general principle often suggested in the course of this volume; that is, *by reducing the weight of the body without impairing the quality of the blood.* But the progress of tricuspid lesions cannot be retarded to the same degree as those of the mitral or even semilunar valves. And, while it is well known that occasional dynamic incompetency of the tricuspid, or slight organic

incompetency is not incompatible with unsuspected health, yet it is no less true that considerable incompetency of this valve is incompatible with assimilation, and with oxygenation of the blood, and hence must prove fatal within a comparatively short period from the moment when it becomes well established.

177. *Fatty degeneration*, admits of postponement within narrow limits, as to its result; but the fatal end will arrive when least expected, and often much sooner than the symptoms would seem to render probable.

Acute pericarditis is seldom fatal. Acute *endocarditis* is somewhat more frequently fatal, but not more so than pneumonia. It almost always leaves disease of the valves, which in the end but perhaps not for half a century, produce death by annulling the function of the heart.

178. *Fibrinous-clots* in the heart almost always cause death in from three hours to as many days.

179. *Ruptures* of the heart, *de natura suarum* are rapidly, though not instantly fatal. *Cardiac dropsies* are curable by means already indicated.

180. *Hypertrophy*, or *hypertrophy with dilatation*, pure and simple, are possibly curable; but such a result is as improbable as that both or either of these lesions can exist without organic disease

of the valves, of the lungs, or of the arteries; or at least without the presence of some external obstruction to the circulation of great and persistent inertia.

Finally: it may be affirmed that after the first chronic lesion of the heart, the others come on—first, compensatory; second, deteriorative; third, compensatory; fourth, more deteriorative; fifth, compensatory; and so on, until its function is annihilated.

Corrollary. The whole object of treatment is to aid nature in these purposes, by diminishing the work the heart has to do, in proportion as its functional ability diminishes, that is, in proportion as its organic lesions advance.

Matter Inadvertently Omitted in Chapter V.

Incompetency and constriction of right auriculo-ventricular orifice.—Incompetency of the *tricuspid* valves is recognized by tolerably certain signs, not difficult of recognition or appreciation. Thus, if there be persistent jugular pulse in all positions of the body, especially the upright, tricuspid incompetency is almost certain. There may or may not be a blowing murmur heard over the tricuspid, (that is, over the sternum between the ends of the fourth interspaces at the right margin of the breast-bone,) such a murmur would be propagated up and down the sternum, in the direction of both the venæ cavæ; but on account of the feeble condition in which the right ventricle usually is, from dilatation, at the period of tricuspid incompetency, the murmur would be inaudible a short distance from its origin; it is, perhaps, never heard at the apex. The murmur is much less frequent than the disease it indicates, because, as already remarked, it is not always audible; and, moreover, it is often marked by a loud regurgitant murmur, as mitral incompetency generally precedes and co-exists with tricuspid incompetency. If the incompetency is considerable, there is generally œdema of the lower extremities, though dropsy is not a

necessary result of any degree of valvular disease. This affection is likely to induce headache from cerebral congestion, and perhaps true softening of the brain, from habitual venous congestion. The *treatment* is that of *dilatation*, and other valvular diseases.

Tricuspid constriction will rarely, if ever, furnish an audible murmur. If heard at all, it should be loudest at the ensiform cartilage, or over the cartilage of the fourth right rib; if associated with tricuspid incompetency, there might be a double murmur. In the present state of knowledge, the *positive* diagnosis of tricuspid constriction is simply impossible. This is not much to be regretted, for it *must* be miraculously rare, and if present, it has no fatal or dangerous significance, unless it be very considerable.

ERRATA.

There are several typographical mistakes, and some errors in syntax in different forms, the proofs of which I did not have an opportunity to revise, but as they do not affect the meaning of the text, they are not referred to in the *errata*.

Omit "backwards," first line, page 22; page 102, for "growth" read growths; page 160, for "shreds of a fleshy column" read shreds of the fleshy columns; page 95, for "hypertrophous dilatation" read hypertrophy with dilatation.

APPENDIX.

A.

¶ 8, page 12. "The first sound of the heart is caused by the closure of the auriculo-ventricular valves, mitral and tricuspid," etc. In asserting this, I mean that the closure of the auriculo-ventricular valves is the *chief* cause of the first sound, and a cause which is no longer debatable, since it has been repeatedly demonstrated as the most efficient, and at least the climax of all the causes of the first sound. But every well-practiced ear will appreciate the beginning of a sound in the systole of the heart, of which the sudden tension of the auriculo-ventricular valves is the complement; thus, in incompetent mitral valves, we appreciate a dull, muffled, indefinite systolic sound, terminated by the murmur of regurgitation, instead of by the click of closure of a perfect mitral valve. This dull, prolonged, muffled, undulatory sound, is, to my mind, undoubtedly the muscular element of the first sound, reinforced by the pressing stroke of the heart against the thorax, and perhaps also by the rush of blood through the aorta and pulmonary artery. I am sure the mechanism and time of the first sound is appreciable, by the duration and force of an indeterminable muffled sound, even when both the tricuspid and mitral valves are incompetent.

B.

a, page 33. "But there is reason to believe its origin—[arrest of the systolic movement of the heart] lies rather in lesion of some portion of the cerebro-spinal axis, than in any

alteration in the structure of the heart itself." I grant this suggestion admits of no positive proof in the present state of knowledge. The lucid experiments of Claude-Bernard have shown that irritation of the pneumogastric nerve slows or arrests the heart's contractions. Now, this is accomplished whether the irritation be applied directly to the pneumogastric nerve, between the heart and brain, or to the roots of the pneumogastric in the lateral tract; or to a branch of the pneumogastric ramifying on the heart itself; or, in short, to any sensitive filament in any part of the body not of the pneumogastric. If the irritation affects a sensitive nerve, it is instantly reflected through the sympathetic upon the heart; and if the sensation be sufficiently violent or painful, the heart is arrested either temporarily or forever; if momentarily, the pulse intermits; if temporarily, the pulse stops several beats; if the arrest is only partial, the pulse hesitates, but goes on. Now, all these phenomena are observed in the variations of the pulse in the different lesions of the heart. Hence I am compelled to modify the suggestion in the text, quoted at the head of this note, and substitute something like the following formula: There is reason to believe the origin of arrest of the systolic movement of the heart lies *sometimes*, in lesions, of the cerebro-spinal-axis—for example, lack of supply of arterial blood to the portion of the encephalon, in which the vagus originates; [in consequence of an atheromatous or ossified artery, etc., etc.,] or some defect in the cerebrum, by which the motor function fails to respond promptly to sensation, or irritation, etc. But it is altogether probable that the cause of arrest of systolic movement, whether temporary or perpetual, may and does often exist in the very substance of the heart itself, or in the quality or quantity of the blood circulating through it, or in the occasional irritation produced by some analogous or heterologous growth in or about the heart. So whether the influence, irritation, sensation or impression, come from the

surface of the body through the sensitive filiments, through the cerebro-spinal axis, or from the internal organs or tissues through the sympathetic, or from the cerebrum encephalon itself, in consequence of an intellection, an emotion, or physical impediment, it is *liable* to cause arrest of the systolic movement, or complete suspension of the heart's action for a moment or forever, according to the nature and force of the influence, etc.

C.

CAUSES OF DROPSY.—Dropsy is caused by something more than mere venous or capillary obstruction. The blood must become deteriorated in albumen before true *dropsy* can take place. Dropsy is a demonstrative evidence of *anemia*. There can be no dropsy without anemia, but there may be excessive *anemia* without dropsy, because the transudation of serum does not take place, even in true hydremia of the blood, unless there is also obstruction to the circulation. Dropsy does not take place until the protein principles (which in the blood are 79 in 1,000,) are reduced to 63 or 58 in 1,000. It will be understood that dropsy is not an *exudation* of inflammation; the dropsical liquid contains no coagulable fibrin, an *exudation* does. The cause of dropsy in Bright's disease is the diminution of albumen in the blood by the albumenuria, in consequence of which the serum charged with still more albumen, is apt to transude, and thus the blood is still further impoverished, not merely by the abstraction of albumen and fibrin in solution, but by the endosmosis chloride of sodium [eight parts of chloride of sodium replace one of albumen] and water for its solution. *The diminution of albumen and increase of salts and water are rigorously coincident facts.* This diminution of albumen in the blood, then, is the true cause of its capability of transudation; and now, if we have any obstruction to the circulation, we have dropsy.

From what is here suggested, it is easily understood why

extreme constriction of the tricuspid orifice may be present and dropsy be absent. Here we may have only one condition of dropsy—obstruction to the venous circulation—while the blood may contain its normal ratio of albumen, and so be incapable of transudation: but once impair this ratio from 75 to 60 in 1,000, and we have dropsy. The integrity of the blood is maintained by respiration and alimentation; obstruct either and we induce a condition favorable to dropsy; hence the necessity of just alimentation, and the freest respiration in chronic diseases of the heart, since these are always obstructive to the circulation.

The true indication of treatment for the cure of dropsy is obvious—restoration of the blood to its normal condition as to albumen, and removal of the obstruction to the circulation. The latter in case of heart disease is impossible in a direct but not in a relative sense. Thus, the weight of the body may be reduced, and so the blood 1 to 8; and in some cases it is practicable to so reduce the quantity of blood, without impairing its quality, that the obstruction may become null. If the blood be restored to its normal quality at the same time, the transudation will be arrested, and the existing dropsy will disappear by the natural secretory functions, stimulated to unusual activity for a few days.

D.

On page 42, I said I would attempt, in a subsequent paragraph, to explain the mode of death in disease of the mitral valve.

It is now well understood that the cause of death in lesions of the *aortic valves* is the lack of supply of arterial blood to the ganglia, both cerebro-spinal and sympathetic, and that the mode of death is by syncope. The immediate *cause* of death in aortic lesion is very well stated by M Mauriac, page 82.

The cause of death in *mitral* lesions is altogether different. Death is here caused by habitual congestions of all the organs behind the mitral valve, the lungs, liver, kidneys, etc. Death, when it occurs immediately, in mitral disease, is always by *apnœa*, and never by *syncope*. The breathing becomes difficult, long before death; the engorged capillaries transude their excess into the cellular tissue, and cause those œdematous infiltrations of the lower extremities so frequent in mitral regurgitation. There are attacks of acute congestion and even of inflammation of the kidneys, attended by albumenuria, by which the blood is stil more permanently deteriorated. As the disease advances, albumenuria becomes more frequent and less curable. Now there is progressive loss of flesh, recurring serous infiltrations (which are removable by purgatives, dry diet, diuretics, expectorants, etc.;) but the struggle of art with an incurable disease cannot last always, and if the patient does not die in a sudden paroxysm of *apnœa*, from pulmonary congestion and bronchial engorgement, he eventually succumbs to the exhausting nature of the disease, and dies from defective alimentation and æration of the blood combined.

For the views expressed on page 67, *et passim*, concerning the cause of pain, I am indebted to "Radcliffe on Pain," etc.

E.

d. page 108. I do not wish to be understood that any considerable effusion into the pericardium would not cause præcordial bulging, but simply that an effusion may be so slight as to cause no appreciable alteration in the contour of the thoracic walls. I have also omitted a sign which is very distinctive of the effusion stage of pericarditis, when the effusion is considerable; that is, the marked increase of triangular dullness in the direction of the apex of the rude triangle represented by the heart and roots of the great vessels.

The apex of this dullness has been known to rise as high as the left clavacle, and the whole triangular dullness to occupy the whole front and lateral portion of the thorax, while the base of the triangular dullness did not fall below its usual place, the lower margin of the sixth rib.

F.

Page 110. "The absent first sound was as accountable on the supposition of effusion, as of softened heart." This has has been objected to, because it is said water is a good conductor, and should transmit the first sound if it were present In answer, I have only to say, that in this case more than half of the first sound was absent by defect of mitral valves; and it is certain that the remaining portion could not be heard as well through eight ounces of effusion as if the effusion were not present; moreover, the absence of the *first sound* in *fatty degeneration* in the last stage is unaccountable if the two sets of auriculo-ventricular valves are competent; that is, the absence of that portion of the first sound caused by the click of the auriculo-ventricular valves; for although from the degeneration of the fleshy columns, on the contraction of which depend the click of the valves, the sound should be diminished, yet if they shut with sufficient force and promptness to resist the resilience of a sound aorta, they should emit a sound as audible at the præcordia, as that proceeding from the same valves when the heart is oppressed with a distended pericardium, and its own dynamism thereby materially diminished, as also by the impediment to the circulation of the blood in its own substance, which must further materially diminish the vigor of the systoles. Hence, the proposition quoted seems to me not inexact.

G.

Page 111. "Saline evacuants," are to be used only when emaciation is still slight, or when with much loss of adipose

and atrophy of muscular tissue, there is considerable dropsy. In this latter condition only temporary benefit can be hoped for, and the salines cannot be used with the same energy and persistence as in the less asthenic dropsies. As a general rule, whenever there is defective alimentation, and hence impaired hematosis, the "warm" aperients or carthartics are preferred to the salines: aloetic, or compound colocynth, pills, or pills of rhubarb, aloes and senna, to which some carminative may be added, are perhaps the best, both in theory and practice. But if the dynamic condition of the patient is such as to tolerate it, (and that of the case mentioned was such an one,) the saline treatment is much the best. A diuretic mixture, composed of infusion of digitalis, acetate of potash, spirits of nitrous ether and cinnamon water, is one of the most active diuretics with which I am acquainted. [We need not exceed a drop an hour of the fluid extract of digitalis.] It must be remembered that diuretics produce less effect on effusions in the pericardium than on cellular infiltrations.

H.

Page 120, ¶ 53. "Embolism from the accidental development of the coagulating principle of the blood." *The coagulating principle* (which is globulin, according to A. Schmidt, who demonstrated it, and whose experiments have been repeatedly verified,) is never developed "accidentally" in point of fact, but is often left undestroyed by accident, and this accident is doubtless the chief cause of *embolism*. *Globulin* is a principle common to all the histologic elements, but it is an integral part of the blood globule itself—hence the superior coagulating energy manifested by blood globules. Blood drawn slowly, coagulates slowly, because the lining of the blood-vessels in its normal state has the power of neutralizing the globulin, else the blood could not remain fluid. It may be stated, then, that so long as the blood is uncon-

taminated with any substance containing free globulin—as, for example, a shred of lymph—and so long as the walls of the vessels are in their normal condition, coagulation of blood in the vessels will not take place, however, slow or frequently interrupted the circulation may become, whether from the action of *veratria*, by which the contractility of the heart is impaired, or from the action of *digitalis*, or *potassium salts*, by which the vagi are probably irritated and the heart's systoles thus retarded, or whether from incompetent mitral valves, by which a systole is wasted, as to its effect in propelling the blood; none of these cause or even tend to cause coagulation of the blood without the presence of the coagulating principle. But directly a blood-vessel becomes diseased in any one spot, say from inflammation so extensive as to cause exudation of lymph on its inner coat, on that point, we have a coagulating principle present, and the production of minute emboli or thrombi begins. So, if there be calcarious deposit in an artery, on a valve, or the endocardium, and the deposit be *not* covered with the healthy lining of the artery, or the healthy endocardium at that point, we have a present negative cause of coagulation of the blood before death—negative, because the action of the living lining of the vessel is necessary to the decomposition of the globulin. *So whenever, from any cause, a portion, small or large, of surface of a vessel, or of the heart in contact with the blood, has lost its vitality—that moment coagulation may begin at that point.* The torrent of the blood may prevent the formation of emboli, and thus secure the patient from harm. Thus, in the living organism, fluidity of the blood is maintained by the healthy vascular walls, and by the simple physical action of the decomposition of the globulin, or its transformation into fibrinogen.

I have objected to giving sulphate of magnesia (page 128, *k*,) because of its tendency when absorbed to induce coagula-

tion of the dissolved fibrin of the blood; thus it should favor the production of *thrombi* in all lax conditions of the system. Of course it would be less injurious in *sthenic* than in *asthenic* inflammations; it is less likely to do harm in large than in small doses, because less likely to be absorbed.

I.

The following tables of Bigot, on the thickness of the walls of the ventricles, from Walshe, are of real practical value. The measurements are in French lines.

MALES.	Base.	Middle.	Apex.
Left Ventricle - - -	1 68–122	5 19–122	3 95–122
Septum - - - -	4 5– 12		
Right Ventricle - -	1 113–122	1 29–244	1 2– 61
FEMALES.	Base.	Middle.	Apex.
Left Ventricle - - -	4 3–8	4 4– 5	3 13– 30
Septum - - - -		4 1–36	
Right Ventricle - -	1 2–3	1 7–24	.673–720

"The mean thickness of the wall of the right auricle has been estimated at about one line French by M. Boulland, that of the left at one line and a half."

Valvular diseases are dangerous to life, and cause annoyance and suffering in the following order:

Tricuspid incompetency.

Mitral constriction and incompetency.

Incompetency of aortic valves.

Pulmonary constriction.

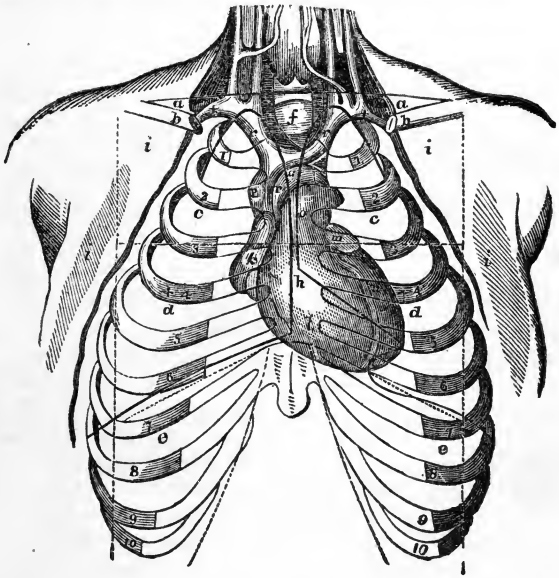
Aortic constriction.

Incompetency of pulmonary orifice and constriction of the tricuspid orifice, are so infrequent, and so little known, that they cannot be estimated as to their effects.

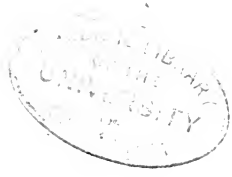
Valvular diseases are not generally causes of sudden death; they are susceptible, as to their effects, of great alleviation, and perhaps to arrest as to their progress. But it must always be borne in mind in estimating the prognosis, that one of them—aortic incompetency—is liable to kill suddenly, and without any marked warning of imminent danger.

The following illustration of the thoracic regions and their contents, copied from Walshe on the Heart, may be found convenient.

DIAGRAM, exhibiting the relationship of the heart and great vessels to the lungs, (in moderate inspiration,) and to the regions of the heart.



1 to 10 inclusive, ribs; *a a* supra-clavicular region; *b b* clavicular; *c c* infra-clavicular; *d d* mammary; *e e* infra-mammary; *f* supra sternal; *g* upper sternal; *h* lower sternal; *i i* integuments turned back; †† nipples; *k* right auricles; *l* right ventricle; *m* left auricle, appendix almost solely seen; *n* left ventricle; *o* pulmonary artery; *p* arch of aorta; the letter *g*, indicating upper sternal region, is also on the aortic arch, transverse portion; *p* (farther to the right) venæ cavæ superior; *r r* innominate veins; *s* innominate artery; *t t* subclavian veins. The dotted lines indicate the outlines of the thoracic regions; the dark lines the edges of the lungs. The heart and vessels are supposed to be full.



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